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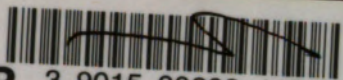
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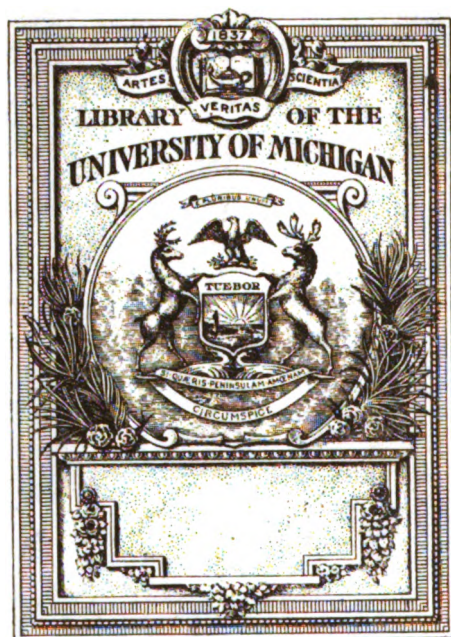
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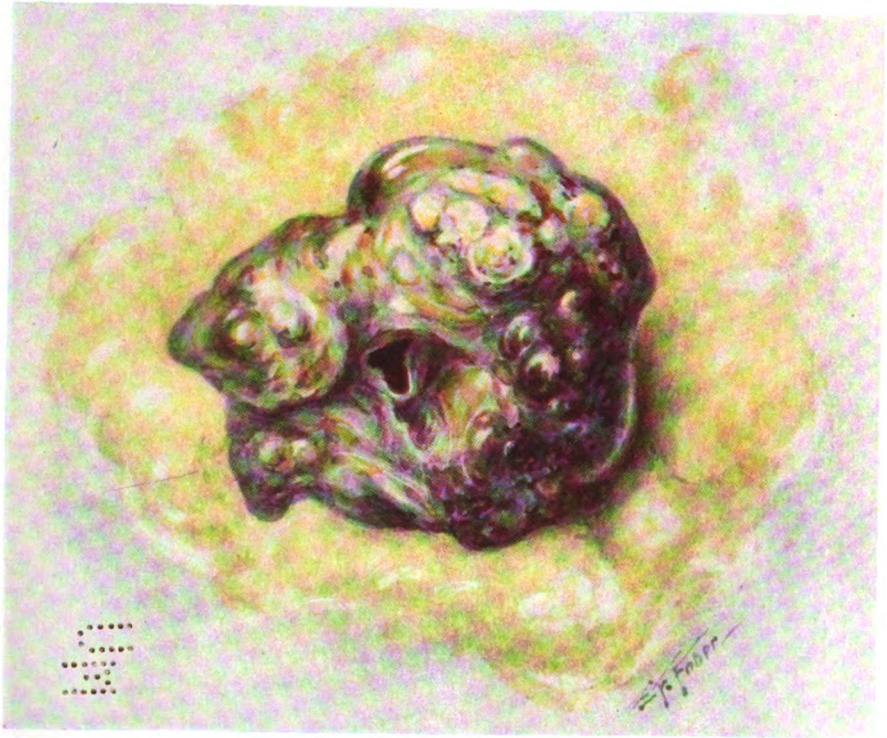


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Pseudomyxoma peritonei.
[See page 188.]

INTERNATIONAL CLINICS

A QUARTERLY

OF

ILLUSTRATED CLINICAL LECTURES AND
ESPECIALLY PREPARED ORIGINAL ARTICLES

ON

TREATMENT, MEDICINE, SURGERY, NEUROLOGY, PÆDIAT-
RICS, OBSTETRICS, GYNÆCOLOGY, ORTHOPÆDICS,
PATHOLOGY, DERMATOLOGY, OPHTHALMOLOGY,
OTOLOGY, RHINOLOGY, LARYNGOLOGY,
HYGIENE, AND OTHER TOPICS OF INTEREST
TO STUDENTS AND PRACTITIONERS

BY LEADING MEMBERS OF THE MEDICAL PROFESSION
THROUGHOUT THE WORLD

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WITH CORRESPONDENTS IN MONTREAL, LONDON, PARIS, BERLIN,
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VOLUME IV. TWENTY-SEVENTH SERIES, 1917

PHILADELPHIA AND LONDON

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Clinics

CLINIC IN MILITARY BONE SURGERY

GIVEN AT THE REQUEST OF THE SURGEON-GENERAL, U. S. A.

BY FRED H. ALBEE, M.D., Sc.D., F.A.C.S., Major M.R.C.

Professor and Director of the Department of Orthopædic Surgery in New York
Post-Graduate Medical School

GENTLEMEN, I am very glad to welcome you here for the purpose of instruction in orthopædic surgery with reference to its application to military requirements. The task of taking up so broad a work in so short a time is a most puzzling one. Orthopædic work as applied to civilian and industrial cases is very broad and varied in character. It has to do not only with bones and joints, but with tendons, muscles, nerves, ligaments, fascia, etc. The competent military orthopædic surgeon will be required to meet all these various conditions as in civil life. They are all more likely to occur under the stress and strain and overwork in the trenches than anywhere else, and in addition to these conditions the orthopædic surgeon will be required to manage a variety of conditions that originate only in war. For this work he must be equipped with the most exact knowledge of anatomy and the physiology of function. We shall therefore endeavor, in this brief course, to use our material primarily for the purpose of teaching fundamentals, and every clinical case has its relationships to fundamental principles.

MAL-UNITED FRACTURE

CASE I.—R. M., aged twenty-four years. Admitted to the Post-Graduate Hospital on October 19, 1917.

Diagnosis.—Mal-united fracture of the lower left tibia and fibula.

The family and personal history of this patient are not important.

Present History.—On April 20, 1917, the patient stepped out on a glass roof and fell through to a store, sixteen feet below. At the time she was six months pregnant, but gestation was uninterrupted by the accident. She was taken to a hospital in Philadelphia and the leg immobilized four different times within four weeks, X-rays being taken at frequent intervals. The cast was finally removed eight weeks after the accident. The patient then started walking, first with crutches and then with a cane, but with a severe limp and much pain. The foot has been in varus since the cast was removed, the varus position being extreme after she began weight-bearing. At the time she came under our observation the varus was extreme, she had a limp on walking, accompanied by extreme pain.

The anteroposterior X-ray of this case shows mal-union in the extreme varus position of an old Pott's fracture (see Figs. 1, 2, and 3). The internal malleolus is displaced inward and has a markedly oblique axis.

The same can be said of the lower end of the fibula, which has become displaced to such a degree that the astragalus has become mortised between the malleoli, which come into close apposition with the sides of the body of the astragalus, thus holding it with the rest of the foot in extreme varus.

The operative correction of this limb had previously been discussed by several consultants. One opinion given was that the deformity should be overcome by means of a correction of the joint. This was to be done by chiselling into the joint through the base of the internal malleolus at the same time that this same procedure was carried out on the external malleolus. The deformity was then to be corrected by changing the plane and relationship of the astragalus with the lower end of the tibia. I believe this would be a mistake in this case as well as in all similar ones, in that we have a joint that has been severely traumatized with a resultant overgrowth of bone and connective tissue which has produced a distinct limitation of motion. This proposed treatment would by necessity increase the traumatism of the joint and therefore would contribute

FIG. 1



Skiagraphs of Case I, showing mal-union and varus deformity, with extreme obliquity of astragalus with tibia.



to additional limitation of motion, to sensitiveness, and to interference with function.

These disadvantages can be nicely avoided in this case by doing the correction of the deformity just above the joint. Such a correction is in every way equal to the one through the joint and eliminates

FIG. 2

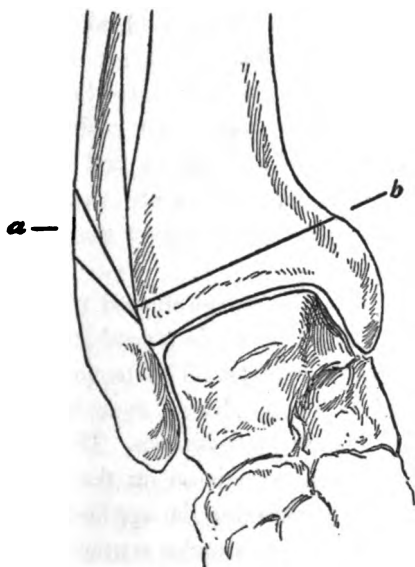


Diagram made from skiagram of Fig. 1, showing mal-union and consequent extreme varus deformity. *a* is wedge-shaped piece of bone removed from fibula, when doing the osteotomy. This space is closed when deformity is corrected. *b* is line of osteotomy of tibia. A wedge cavity between the tibial fragments is opened at this point when the deformity is corrected and the wedge-graft from the fibula at *a* is inserted at this point.

FIG. 3

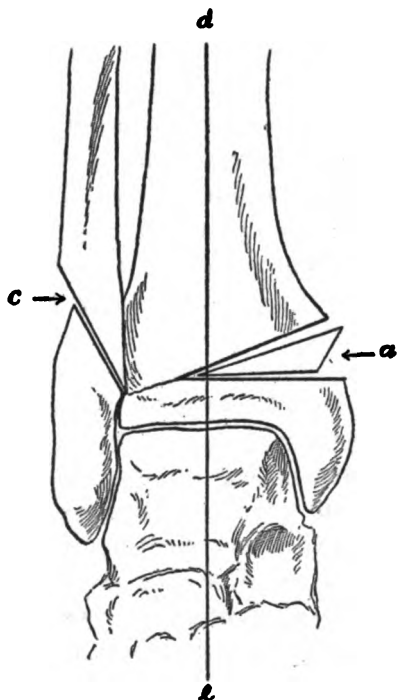


Diagram made from skiagram taken after operation correction. *a* is the fibula graft placed into cuneiform cavity between inner side of tibial fragments after correction of deformity shown in Figs. 1 and 2. The cavity at c.m. fibula is at the same time completely closed by the correction. The line *d* indicates the ideal restoration of alignment accomplished.

the disadvantages above enumerated of additional traumatization to the joint.

Operation.—We will first make a linear incision over and parallel to the lower end of the fibula and through the periosteum on its outer aspect. The periosteum is separated laterally by means of a sharp

periosteal elevator about the whole lateral diameter of the fibula just above the old point of mal-union. By means of my motor saw and a sharp, thin osteotome converging cuts are made obliquely downward to a point about one-half an inch above the astragalotibial joint (see *a*, Fig. 2). A long, slender wedge of bone is thus removed and placed in normal saline for future use. The wound is then packed with sponge compresses soaked in warm saline. The next step is to lay bare the lower end of the tibia on the internal aspect in like manner. Using my motor saw and a sharp, thin osteotome, the lower end of the tibia at a point about three-fourths of an inch above the ankle-joint is severed at right angles to its long axis (see Fig. 2, at *b*). The varus and lateral deformity of the ankle are then corrected by manual force. This automatically closes the wedge cavity in the lower end of the fibula (see Fig. 3 at *c*) and at the same time produces a wedge cavity of approximately the same size between the tibial fragments at their inner aspect (see Fig. 3 at *a*).

While the foot is held in this position the wedge-shaped piece of bone removed from the fibula and placed in saline is placed into the wedge-shaped cavity between the tibial fragments. The periosteum, which is strong and considerably thickened, is drawn over it with medium strands of kangaroo tendon, interrupted sutures. The skin is closed by continuous sutures, and the skin incision on the fibula side is closed in like manner. A gauze dressing is applied with sterile absorbent cotton placed over it. I always make a practice of placing the sterile absorbent cotton over the gauze dressing of any wound involving bone where there is likely to be any amount of exudate or bleeding.

The rationale is this: It is well known that the gauze dressing easily becomes saturated with blood or exudate, and as soon as saturation takes place the dressing is no longer impervious to penetration by bacteria. The absorbent cotton, on account of its compact nature, is not readily penetrated by the exudate or blood, and therefore, as a matter of security, I always place the absorbent cotton over the gauze dressing and before I adjust the non-sterile plaster, cotton, or flannel.

With the foot held carefully—and I would emphasize *carefully*, because holding the foot of such a case is more important than the

application of the dressing—we now apply the plaster-of-Paris dressing from the toes to a point one-third way up the thigh, with the knee flexed at twenty degrees.

The object of placing the plaster above the knee with the knee flexed is to prevent inward or outward rotation of the foot upon the leg. I wish to call attention to the fact that I have completely severed both the tibia and fibula at a point just above the ankle-joint, and therefore the dangers of rotation of the foot are imminent.

In this connection I would like to say that in many clinics a plaster dressing to fix the foot in similar cases is limited to the tubercle of the tibia, and the danger of rotation inward or outward is prevented only by friction of the plaster cotton with the skin of the leg. I believe this is very untrustworthy, because it is well known that we have an immediate shrinkage of the diameter of the leg on account of atrophy from disuse. There will also be a packing of the cotton at the same time, and both of these influences will produce a rapidly increasing looseness of the plaster on the leg and a corresponding decreasing or diminishing resistance to rotation of the foot on the leg. I wish also to call attention to the fact that we have left the tourniquet on until the plaster dressing was completed. This is a rule I always follow; that is, I apply the dressing and the fixation dressing to an extremity before removing the tourniquet. In preparing the patient for the operation I applied the tourniquet on the thigh as you saw. On account of the tendency of the old deformity to relapse we will leave this cast on five weeks, after which time passive motion and massage will be employed in order to get back the full amount of function.

TUBERCULOSIS OF HIP-JOINT

CASE II.—C. B., aged thirty-three years, a clerk by occupation.

Diagnosis.—Tuberculosis of the left hip.

Family History.—There is no tuberculosis in the family.

Personal History.—At the age of nine years this patient fell from a horse and thinks he dislocated his hip at that time. Later in the same year he had a fall on the left leg. He has had no long illnesses except the present one.

Present History.—The patient has walked lame since the age of nine years, and attributes his lameness to the falls recorded above. At the age of twelve years a flexion deformity developed at the knee. Traction was applied and the limb immobilized for nine months, after which he wore a brace and walked on crutches for two years.

At the age of fifteen years he had an abscess at the external surface of the hip. This abscess was operated on by three incisions, in an Allegheny hospital, in 1899, and again opened by a surgeon in New York. He removed dead bone and curetted the cavity. After that time the patient had no trouble until 1914, when he began having neuralgic pains. In December, 1915, he developed a gluteal abscess, which has never healed since that time and which has been followed by two other spontaneous ruptures in front and behind the hip-joint which have persisted to date.

The physical examination, in conjunction with a study of the X-ray plates, reveals an old arthritis of the hip, undoubtedly of tuberculous origin, with complete bony ankylosis. The leg has atrophied, and there are four sinuses distributed about the lateral aspects of the hip. The passage of the probe shows that they lead toward the hip-joint. The limb is in a position of about twenty degrees flexion and about fifteen degrees adduction. The X-ray, in addition to showing bony ankylosis, shows an enlargement of the diameters of the neck of the femur, with increased density. By means of an ordinary incision and curettage it is most difficult to reach the remote recesses in the bone and find the sequestra in such a case.

This statement is emphasized by the fact that this man has been operated upon three times without success. It will be noted that this patient does not complain of pain in his hip, for the reason that his hip is firmly ankylosed.

Operation.—In this case, in order to expose thoroughly the bone-pockets about the hip, I will not attempt to follow up the individual sinuses, all of which lead toward the hip-joint, but will expose the whole region of the hip by means of the Smith-Peterson approach. I will make the incision along the outer border of the sartorius down from the anterosuperior spine for a distance of about four inches. This incision is then carried from the anterior spine posteriorly

along the iliac crest for a distance of three and one-half inches. By means of an osteotome all of the muscles are turned downward and backward from the outer surface of the ilium. At the same time, by blunt dissection through the muscle planes between the sartorius anteriorly and tensor fascia femoris posteriorly, the hip is approached anteriorly.

This, you see, gives an excellent exposure of the anterior, lateral, and posterior aspects of the hip-joint. We will now proceed to explore the sequestra and pockets by means of a grooved director, and by means of a strong curette and bone-gouge we will transform these pockets into bone depressions without overhanging walls. I am taking the greatest care to trace the bone-pockets to their remotest recesses. These recesses are now wiped out with tincture of iodine. The chronic inflammatory sinuses in the skin and subcutaneous tissues are now thoroughly excised. We are now ready to close the wound by suturing the upper border of the muscles back to the crest of the ilium by means of a continuous suture of catgut No. 1, placed through the fascial investment at the crest of the ilium and the upper border of the muscles. The skin is closed by continuous sutures of zero plain catgut. The sinuses are now packed with iodoform gauze.

I wish to call attention to the fact that the sinuses in this case have persisted because of secondary pyogenic infection with sequestration and death of the bone. When sequestration has taken place subterranean curettage is a very untrustworthy technic. The complete exposure, with most careful search for dead bone, is the only procedure which offers a prospect of a reasonable percentage of successes.

I wish to instruct the interne to begin removing the drains at the end of twenty-four hours. The drains are to be gradually withdrawn until they are completely removed. After this the drains are to be placed more superficially and tightly packed in the skin to prevent the cavities from closing too rapidly. The deformity will be corrected at a future operation and not until the wounds are entirely healed.

INFANTILE PARALYSIS

CASE III.—G. G., aged six years. American parentage.

Diagnosis.—Paralysis of the extensors of the right leg resulting from infantile paralysis.

Family History.—This is unimportant.

Personal History.—The patient has had measles and mumps.

Present History.—The child has a deformity of the right leg and foot following an attack of infantile paralysis sixteen months ago. The extensor muscles of the right leg are paralyzed, in consequence of which the child has been unable to walk correctly, and stumbles and falls easily.

Physical Examination.—The thigh is much atrophied and there is a slight knock-knee. The quadriceps group of muscles is entirely paralyzed. If I place the child in a sitting posture on the table, with the leg hanging over the edge of the table at right angles to the thigh, you can see that the child is unable to swing the foot forward and upward at all. Palpation of the quadriceps group of muscles during the child's efforts also reveals that there is no evidence of muscle contractility. We will now place the child flat on the table with the face downward and with the knee and foot resting on the table. You see when the child is asked to flex the leg at the knee she has excellent strength in the hamstring muscles; in fact, all of them seem to have a normal amount of strength. We will test for the strength of the biceps femoris, as this is by all means the best of the hamstring muscles to transplant forward to take the place of the quadriceps group. The biceps is apparently of normal strength. We have informed the family of these facts and have advised an operation by which the biceps will be transplanted into the patella. This is the operation which we will now perform.

Operation.—With the child under ether the leg is elevated so that it is at right angles to the trunk. It is held in this position for a few minutes and the blood milked toward the hip. A tourniquet is then placed as high up on the thigh as possible. It is important in tendon transplantations as well as in bone transplantations to avoid traumatization of the transplant material. Therefore in this case I will prepare the site of the insertion of the muscle before I expose the portion to be transplanted. Through an incision about one and one-half inches in length I am now exposing the upper

and outer corner of the anterior aspect of the patella. I would like to call attention to the fact that this is a very vascular region, and if we do not use a tourniquet we will have a great deal of bleeding.

With a scalpel I will now cut through the soft tissues enveloping the patella at about the junction of the middle and outer one-third of the anterior surface of the patella at its upper border. Now with a sharp, thin osteotome I am turning up small hinge-doors of bone and periosteum under and between which we propose a little later to insert the biceps tendon. This wound is now packed with saline gauze compresses to prevent drying. The biceps femoris is now laid bare by an incision extending from the head of the fibula upward over the thigh for eight or ten inches, and this muscle is developed, taking care not to traumatize or cut either of its surfaces. The tendon is developed at its insertion into the head of the fibula and just above this point where it comes into relationship with the external popliteal nerve and the lateral ligament, which is round in contour and lies beneath and a little posterior to it. I will now separate the tendon from the head of the fibula by taking out a wedge-shaped piece of the head of the fibula itself with the insertion of the tendon. With a special forceps devised so as not to crush, the end of the tendon is seized and drawn upward and outward while I dissect the muscle off the linea aspera to the extent of about two-thirds of its attachment. During this procedure you see the assistant is dropping saline on the muscle from a sponge held above the wound. This is to keep it from drying. By means of a pair of long, curved scissors I will now tunnel obliquely through the subcutaneous tissues from the patella upward in the line the transplanted muscle is to take. You will see that by opening the scissors we are making the tunnel very large, so that it will receive without constriction the lower part of the belly of this large muscle. I will now place this long Kelly forceps through the tunnel from the patellar side and grasp the bony insertion in the end of the tendon and pull it through the tunnel to the patella. By means of fine kangaroo tendon on a strong cervix needle we will now firmly suture the bony insertion of this tendon under the hinge-doors of bone on the anterior surface of the patella which we have previously prepared.

I wish to call attention to the fact that I am using a continuous over-and-over suture rather than interrupted sutures, the object being not to trust to knots and always to avoid knots in the tissues at this point. The over-and-over suture is much more trustworthy. I wish to call attention also to the fact that I am using an absorbable suture and not silk. I believe it is important never to use a foreign body in and about bone if it can be avoided. In this connection I always think of Mr. Jones's experience of finding streptococci in silk with which he had previously anchored a tendon, though no symptoms had presented themselves.

I am now closing both incisions with continuous sutures of zero plain catgut. We will now apply a plaster cast from the toes to the groin, removing the tourniquet afterwards.

In a case of this kind I instruct the interne to keep the leg elevated on two pillows. The plaster cast will be removed in three weeks, after which a brace or posterior splint will be worn to prevent the knee from flexing. This will be continued for a couple of months, when all apparatus will be removed. In such cases as this normal function is restored.

Muscle Training.—I wish to go on record as strongly in favor of muscle training, as it is of the greatest service in transplant cases. The results in these cases are very gratifying indeed.

Subsequent History.—This patient is able to extend his leg or raise his foot and is very much pleased, after the removal of the splint, to find that he has the power to flex his leg.

RECURRENT SUBLUXATION OF THE PATELLA

CASE IV.—A. D., aged twenty-three years.

Diagnosis.—Recurrent subluxation of the left patella.

The *family history* is unimportant and the patient was never sick before.

Present History.—In 1910 or 1911 the patient had a fall from a horse, which was followed by "water on the knee." She did not think it was of much consequence and remained in bed only two days. A year later, while standing and attempting to turn around, the patella slipped out, throwing her. She put the patella back manually. Once since that time while playing tennis and once when

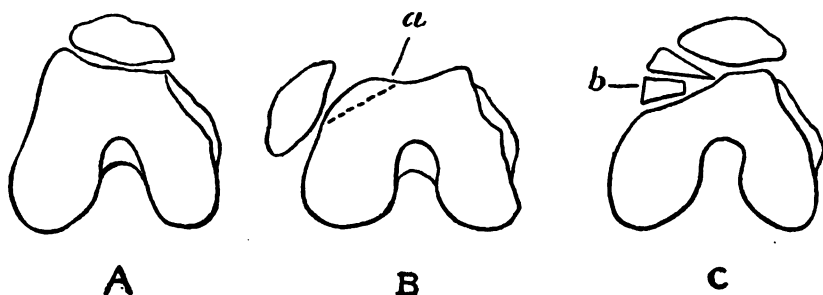
turning around the same accident was repeated. After undue exercise, even when there was no subluxation, the patient has had a constant ache in the knee, due apparently to the patella constantly riding up on the outer condyle of femur. I have never seen a patient who was in a more perturbed state of mind concerning an ailment than this young woman. She is in constant terror lest the patella slip out of place. She has not dared flex her leg, and it interferes with all her pleasures. She can not dance or enter into any athletic exercises whatsoever. I had great difficulty in persuading her to allow me to examine the knee at all, because she was afraid of the patella slipping out because of my manipulations.

A great many types of treatment have been suggested for this condition, and it is almost unanimously agreed that all conservative brace treatment is of no avail, and the same is true of many of the operations that have been relied upon. In many of these operations connective tissue is relied upon to withstand a constant tension over long periods of time—an unreliable procedure to begin with. In such type of operation there is reefing of the inner part of the capsule. Another operation is to transplant one hamstring into the inner side of the patella so that its contraction will hold the patella inward and prevent its outward displacement. This is not a trustworthy operation, because it necessitates a transplanted tendon pulling around a corner, which is sure to develop adhesions. Another operation is that which changes the location of the ligamentum patellæ by either changing the bony insertion *in toto*, internally, or by splitting the ligamentum patellæ and drawing the outer half beneath the inner half and anchoring it on the inner side of the tip of the tibia. Still another operative procedure described in the literature is to do a complete supracondylar osteotomy of the femur and turn the external condyle forward. Elevating the external condyle forward is certainly desirable, but this procedure, at the same time that it accomplishes this, also changes the line of the quadriceps pull in that its relation to the tubercle of the tibia is changed. The magnitude of this operation is also to be considered. A more recent operation is to increase the intracondylar gutter by chiselling away a large amount of bone with its investing joint cartilage and synovial membrane, and then use a transplanted fascial flap to cover over this

bare surface so that the patella will not adhere. This operation, with all its chances of interfering with free motion, does not supply the essential mechanical conditions, namely, elevation of the external condyle anteriorly without changing the line of quadriceps pull (see Fig. 4, A, B, and C).

Operation.—The operation I am about to do I devised some five years ago, and published it at about that time and in my book on "Bone-graft Surgery." Brackett also did a somewhat similar operation at about the same time. But, to recapitulate, the essential condition to establish here is to elevate the anterior external condyle of the femur, thus increasing the depth of the condylar groove and preventing external displacement of the patella. This can be sim-

FIG. 4



A indicates the normal size and anterior shelving of the external condyle, which prevents in the normal knee lateral displacement of the patella. B indicates the flattened external condyle with a consequent luxation of the patella outward, and the proposed osteotome cut at a. C indicates anterior lifting of the condyle to block the recurrence of the dislocation of the patella with the wedge tibial graft b in place.

plest and best done by placing a tibial bone-graft wedge beneath the external condyle after this has been elevated by osteotomy. I will now make an incision over the outer condyle of the femur down to a point about an inch posterior to its anterior articular border.

We find here that we do not go through the joint space, as the soft parts are attached to the femoral condyle at this point. I will now introduce an osteotome and by an incision through the outer side of the condyle, obliquely forward and inward (see Fig. 4 at a), I will now elevate the condylar fragment forward, producing a wide cavity, as is shown in the diagram (Fig. 4C at b). The osteotome is now withdrawn and the wound packed with saline compresses.

The anterior-internal surface of the tibia is next exposed by a circular cut of the skin and subcutaneous tissues. A wedge-shaped pattern of a size to give a proper elevation of the condyle of the femur is mapped with the scalpel on the anterior-internal surface of the tibia. By means of my motor saw we will now remove a wedge of bone about one and one-half inches in length and consisting of the full thickness of the cortex of the tibia at the lower one-third. We choose the lower one-third because the cortex is thicker at that point. The graft should also include the periosteum on one side and the endosteum and all the marrow that will cling to the other side. This graft is now drilled with a very small drill in the central portion. It is now placed in the wedge-shaped crevice at the outer side of the condyle of the femur (see Fig. 4C at *b*, Fig. 5 at *a*, and Fig. 6 at *a*), the wedge action being used to elevate the condyle anteriorly (see Fig. 5 at *c*). The periosteal surface is outermost and flush with the sides of the femoral condyle. With the small motor drill I am now making holes in the sides of the condyle obliquely to meet the holes in the tibial graft both from above and below. A medium strand of kangaroo tendon is then placed by means of a curved needle of proper curve through these holes and tied (see Fig 5 at *b*). Additional sutures of the same material are placed in the periosteal structures above and below. Both wounds are closed with continuous sutures of No. 1 plain catgut. The dressing and plaster splint are applied up to the point of the tourniquet, which is removed and the plaster extended to the groin.

Postoperative Care.—The plaster splint will be removed at the end of four weeks, when a posterior splint will be applied to prevent bending the knee and the patient will then be allowed to walk. All splints and apparatus will be removed at the end of six weeks after the operation.

UNUNITED FRACTURE

CASE V.—J. C., aged twenty-eight years. Occupation, lease man.

Diagnosis.—Ununited fracture of the left tibia.

The family and personal history are unimportant.

Present History.—On February 25, 1916, the patient was riding in an automobile which was hit by a train. He was etherized the

same day and the fracture set. Five days later it was set again under ether. He then wore a plaster-of-Paris cast for four months. Six weeks after receiving the injury he sustained a fall which did not break the cast.

Operation.—For the repair of this lesion I am going to use either a sliding bone-graft from the upper fragment or a graft removed from the other tibia. I believe in this case a sliding graft from the same tibia will serve our purpose, but I shall reserve my final decision until the bone is exposed and the exact condition determined.

We are now exposing the point of fracture of the tibial fragments on either side by a grooved incision and turning back the skin and subcutaneous structures. The incision is so placed that when we close it, later on, the line of sutures will not come over the graft. This is a general rule in bone-graft procedures which should never be violated; that is, the skin incision should never be so placed that when the wound is closed it will come over the gutter from which the graft was removed or over the graft itself, as it is important that these regions be thoroughly sealed from communication with the exterior.

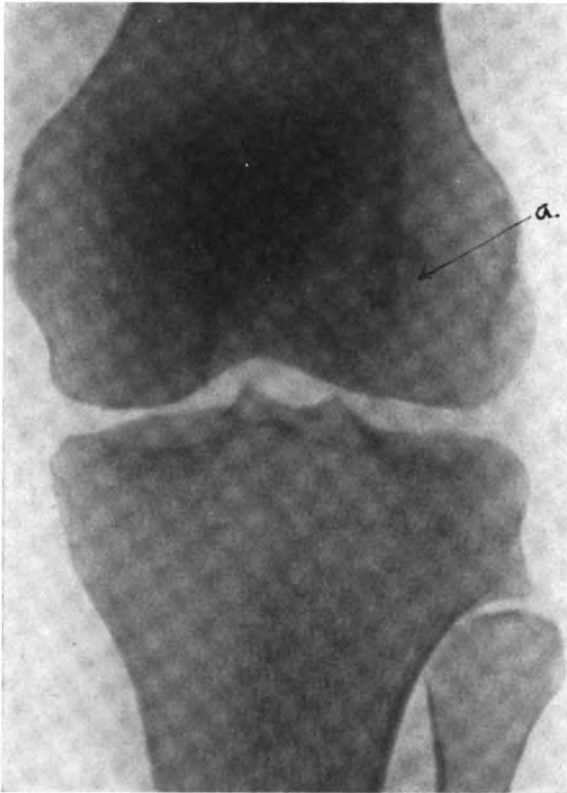
We will now split the periosteum on the lower fragment downward from the point of non-union for two and one-half inches. We will next peel this laterally and remove the fibrous union from between the tibial fragments. I wish to remark that the tibial fragments are in very good end-to-end apposition and the fibula is united. It is difficult to say why this man's tibia did not unite. He is in good health and the blood tests reveal no systemic disease. Whether there was soft tissue, such as muscle, between the fragments following the fracture we can not determine at this time. I have operated on many just such cases as this in which non-union could not be explained. In my experience the frequent causes of non-union may be briefly enumerated: (1) Compound and comminuted fractures with extensive removal of bone. I had the privilege of observing many such cases in France a few months ago, and therefore I shall advise you very strongly never to remove fragments in such cases if you can avoid it. (2) Severe infection following such cases is also a prolific cause of non-union, and syphilis likewise causes a certain number of these cases. (3) Last,

FIG. 5



Skiagram and diagram of skiagram of Case IV, showing tibia wedge-graft *a* in position under the elevated femoral condyle *c*. The kangaroo tendon ligature in drill holes in graft and femoral condyle above and below graft is shown at *b*.

FIG. 6



Anterior-posterior skiagram and diagram from it of Case IV, showing the tibia wedge-graft *a* in place under anterior fragment of condyle, which has been elevated.

but not least, Lane's plate when applied by faulty technic is a cause of non-union.

To proceed to our operation—we find that there is every evidence that the bone in the upper fragment is of good quality and suitable for grafting; we will therefore refrain from separating its periosteum and will map out the size of our graft. Using the twin saw as calipers, we take the maximum measure of the marrow cavity of the smallest diameter of the tibia where we are to make our gutter; this point comes in the lower fragment. Now that we have determined how wide we can make the graft, we will lock the twin saw by turning the proximal saw from us. With the twin saw as a guide we will map out with the scalpel on the periosteum the pattern of our proposed sliding graft. With the twin saw placed in the motor we now make parallel cuts for a distance of two inches below the non-union in the lower fragment and four inches above it in the upper fragment, following the pattern laid out over the marrow cavity. We next remove a strip of bone by the same method from the lower fragment. We cut off the ends of these strips of bone with our small rotary saw. Please notice that we always take out the cutting tool before placing our motor back on the instrument table. This is an important rule which should never be violated. The surgeon should become so accustomed to taking his tools out of the motor immediately upon completing a manœuvre, even if he must replace it in a few seconds, that it is second nature to him; this will obviate any possibility of accident, since the motor is perfectly harmless so long as there is no cutting tool in it.

By means of the osteotome we will now remove a strip of bone from the lower and upper fragments, great care being observed not to crack or break the proposed sliding graft from the upper fragment. This can best be done by driving the osteotome in as a wedge by successive gentle blows of the mallet along both parallel saw cuts. When it is observed that the graft will easily wedge sidewise it is safe to drive the osteotome under its end at the point of non-union. I wish to call attention to the plug of sclerosed bone which fills the entire cavity for a distance of approximately one and one-half inches from the point of non-union. We must remove this to a sufficient depth to allow the sliding graft, with all the marrow that will cling to it, to fit into place so that its periosteum will be flush

with both posterior fragments. You will note that on either side of this sclerosed bone the marrow cavity is laid bare with its vascular content. We now drill holes in each side of the gutter for fixation kangaroo ligatures. These are drawn through by means of the Reverdin threader. We now pull up the kangaroo loops from the bottom of the gutter and place the needle-holder beneath them. The graft is now placed beneath the needle-holder under all the loops and the needle-holder is withdrawn. The graft is now very gently dropped into its gutter, with one-half in the lower fragment and one-half in the upper fragment. The kangaroo ligatures are tied over it, and the second one-half knot of each ligature is locked by tying it about a ligature of No. 1 chromic catgut at right angles. The strip of bone removed from the lower fragment is now split into three slivers, one of which we will cut into small fragments or "bone seed" and will place in the spaces between the tibial fragments around the inlaid sliding graft to fill in the space left by the removal of the fibrous union. The remaining two slivers we will place on the external and internal sides of the point of non-union beneath the periosteum, one-half of the sliver in contact with the upper and one-half in contact with the lower fragment. The subcutaneous structures, including the periosteum of the lower fragment, are now approximated by means of a continuous suture of chromic catgut. We close the skin by continuous suture of zero plain catgut, and we place the limb in a plaster-of-Paris cast from the toes to the mid-thigh region.

Postoperative Convalescence.—This cast will be removed at the end of eight weeks. The additional duration of support will be determined chiefly by means of the X-ray study of the amalgamation of the graft and callous formation.

I wish to emphasize at this juncture the importance of doing this type of work by the most accurate cabinet-maker's technic. This technic is no longer difficult of execution, since by necessity I was compelled to devise a motor outfit with the twin saws which you have just seen at work. In other words, this is machine work. The twin saw is an automatic tool which we use to make a gutter and to remove a graft for that gutter. It insures a perfect and uniform fit.

FIG. 7



Anterior-posterior and lateral views of Case VI, showing the degenerative and cystic condition at lower end of tibia.

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CHRONIC OSTEOMYELITIS

CASE VI.—E. L., aged two and one-half years.

Family History.—The family history is negative, except that there may be some question as to whether the father might not have had tuberculosis. There are six other children in the family, all in good health.

Personal History.—There is no history of injury. When the child was about six months old the mother noticed that the child apparently had pain in his ankle. This was soon followed by swelling. The mother then took the child to a doctor, who expressed the opinion that the condition was tuberculous. He put on a plaster cast, which he continued for nine months, but the patient did not do well. The swelling increased and the child continued to complain of pain. The swelling began to progress upward along the leg. The child then came under my observation.

Physical examination shows that the child has a pronounced limp, and in the region of the right ankle a marked swelling extending three inches above the ankle. There was tenderness on deep palpation. Passive motion caused some pain, but not so severe as one would have expected with acute tuberculosis.

Now the problem that presents itself here is one of diagnosis. We will have to trust to the X-ray largely in settling this question. Several X-rays have been taken (see Fig. 7). They show a large cavity, one and three-quarters inches in length and seven-eighths of an inch in breadth, in the extreme lower end of the tibia, apparently extending into the epiphysis, with a certain amount of disintegration of the epiphyseal cartilage at the central portion. There is moderate induration of the bone about this cavity, with no evidence of breaking through to the periphery of the lesion. From the X-ray and the physical examination it is evident that the diagnosis still remains doubtful. The conditions to be considered in arriving at a diagnosis in this case are the following: bone cyst, osteitis fibrosa, osteomyelitis hemorrhagica, quiescent stage of Brody's abscess, osteomyelitis with a low grade of infection, or new-growth. Operation, however, is indicated.

Operation.—We will first place a tourniquet above the knee and make an incision over the anterior-internal aspect of the lower portion

of the tibia down to the periosteum. Then, with a circular rotary electric motor saw, a trap-door of cortical bone is cut and turned outward laterally, with the periosteal structures as a hinge (see Fig. 8). A definite circumscribed cavity in the bone, the shape of a hen's egg, is thus disclosed. This cavity is filled with fluid in which are three bone sequestra and a large amount of yellow, cheesy material. We now thoroughly clean out the cavity, and find that it extends completely through the epiphyseal cartilage and almost through the

FIG. 8

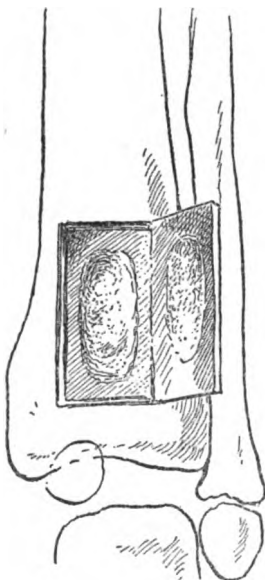


Diagram indicating hinge door of bone cortex turned up upon the periosteum and periosteus structures as a hinge in Case VI. It also indicates the definitely circumscribed cavity beneath which was filled with Mosetig-Moorhof bone-plugging material.

epiphysis of the tibia. The upper surface of the synovial membrane is seen at the lower end of the cavity. When the ankle is moved the joint motion can be seen through the synovial membrane. We now carefully dry the cavity and with a glass syringe completely fill it with Mosetig-Moorhof bone-plugging material, a few degrees above body temperature. The trap-door of cortical bone is now turned back into place and fastened with interrupted sutures of fine kangaroo tendon. The soft tissues are drawn together by means of zero chromic catgut. The skin incision is closed with zero plain

catgut. The limb is now placed in a plaster-of-Paris dressing from the toes to just above the knee.

Doctor Meeker's Pathological Report.—The gross specimen comprises several soft pieces of tissue, some granulation tissue, and some semi-necrotic caseous material. One piece contains bony spicules. Another piece is a thin slab of bone, necrotic and hemorrhagic on one side, with some tabs of granulation tissue. The microscopic examination shows in the soft parts granulation tissue with numerous points of bone dotted about. In this fibrous tissue formation are giant-cells of a foreign body type and many plasma-cells. The giant-cells differ from those of tuberculosis in that the nuclei are fewer, larger, and centrally located. In the decalcified bone sections a large amount of fibrous tissue is seen between the trabeculæ, replacing the bone-marrow. In other areas there is rarefaction of bone. In other words, vascular granulation tissue is extending at the expense of bone. Kaufmann describes a rarefying osteitis near the epiphyses with cyst formation and granulation tissue with giant-cells. It may very well be a congenital failure of bone formation (see Kaufmann). It is the lesion of rickets or osteomalacia.

Diagnosis.—Chronic osteitis or osteomyelitis of obscure origin.

Subsequent History.—This child made a good recovery. The skin healed by primary union and there has been a complete cessation of pain.

CLINICAL LECTURE

AT UNIVERSITY HOSPITAL, PHILADELPHIA, OCTOBER 24, 1917

BY DR. G. G. DAVIS

CONGENITAL LUXATION OF THE HIP

THIS little girl, who is four years old, has a congenital luxation of the right hip. It was replaced, but did not stay in place. Some of these cases show a tendency to slide toward the upper edge of the acetabulum, and it is very difficult to keep them down low enough. When we attempt to keep them in their place by putting the leg at right angles to the long axis of the body, in the typical Lorenz position, the head of the femur will slide a little bit up, and often this is not noticed for quite a while. Then, after the lapse of a certain length of time, when you take off the plaster of Paris, you will find that the head is higher than it ought to be. That is what occurred here.

In this case we removed the plaster and placed the limb in the position of greatest flexion, so as to bring the knee up toward the axilla, it still being out from the body in the coronal plane, and put it up in that position (Fig. 1). It is necessary to put on such a dressing that will keep the knee flexed, but not to have it horizontal when the child is standing, but placed at an angle upward of approximately 45 degrees. We keep it there for one month, two months, or three months, and then gradually bring it down to a right angle; and finally, after another one, two, or three months, bring it down slowly the remainder of the way.

This is illustrative of one of the difficulties that we encounter in the treatment of these congenital luxations. If you had a traumatic luxation, you would not experience such a difficulty; for if it were a pure luxation not complicated with fracture, we would have a good acetabulum, a distinct cup. If you once succeed, in a traumatic luxation of the hip, in bringing the head into the acetabulum, it will stay there, in spite of all motions except one—that of abduction. In a traumatic luxation, if you wish to luxate the bones of any joint, you abduct or hyperextend it. Abduction is only another name for

hyperextension of certain joints. You speak of abduction of the hip and abduction of the shoulder, but of hyperextension of hinge-joints. Therefore you will observe that if we wish to dislocate a plain hinge-joint we hyperextend it. When a person is thrown forward and strikes on the thumb it hyperextends it, and you get that troublesome condition, metacarpophalangeal dislocation of the thumb. When you come to the elbow, if a person is thrown down in such a way that his forearm becomes fixed, and he is then pushed backward, the elbow gets hyperextended. That is a common way for luxation of the elbow to be produced. If you wish to dislocate the shoulder you hyperabduct it, and the bone comes out of the socket.

When you come to the lower extremity, the phalanges are dislo-

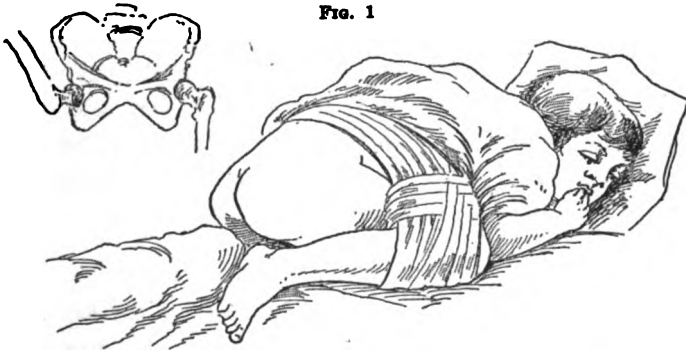


FIG. 1

Leg held in corrected position in congenital luxation when the head of the femur tends to go too high.

cated by hyperextension. When a person jumps out of a carriage and strikes the ground, the phalanges will be bent back on the dorsum and you get a luxation of the phalanx on the end of the metatarsal bone. It is the same if you hyperextend the ankle. The foot bones slide backward and the leg bones slide forward. To dislocate the knee—hyperextend it—it may occur in this way: A person, in jumping from a train, may have the leg go into a hole and held firm while the body is carried forward and the knee bent forward. In the hip all you have to do is to abduct it sufficiently to rip the capsule and give it a twist, and out goes the head. Rupture of the capsule is performed by simple abduction. If you want to dislocate the jaw, you simply extend it or open it too widely. We see, therefore, that the production of all the luxations is governed by one principle—that of hyperextension.

This case shows how in some of these congenital luxations the acetabulum is not well formed. The cup is not distinct. The cotyloid ligaments are flat and the capsular ligaments are stretched, and there is nothing to hold the bones in place except the position of the extremity and the tension of the muscles. Sometimes the ligaments on the anterior portion of the joint also help.

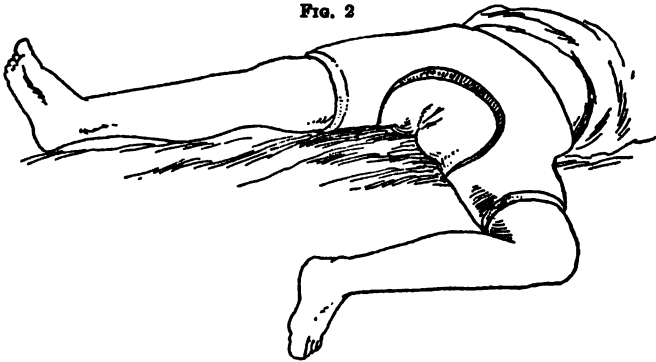
In the X-ray is shown the cartilage which marks the middle of the acetabulum, in children, and one can see how this extremely flexed position brings the head directly opposite the desired spot. In between the shadows of the bone on each side of the joint line is a space which is filled with cartilage. Inasmuch as the X-rays pass through cartilage without throwing a shadow, you can not always determine the exact form of the socket from the X-ray plate. You can, however, usually form some idea of the shape of the acetabulum from the bony outline. On one side there appears to be a nice shelf, but you can see that on this other side the shelf is not marked; but, while the bone shadow is not so marked as that of the other side, the rim or shelf may be eked out by a triangular piece of cartilage—in which case the head may stay in its place just as well as on the other side.

SECOND CASE

This is another case of *congenital luxation of the hip* that I reduced last Wednesday. I am rather interested in this way of putting up a luxation in plaster of Paris after reduction. Try to determine by palpation whether the head is in its proper place, beneath the femoral artery, close to the middle of Poupart's ligament. Note how much lower the head is than the anterior spine. By putting one finger of the left hand on the head in front and another finger of the right hand on the trochanter behind and raising it a little you can balance the head between the two and feel it move and thus be sure of its position. In putting up these congenitally luxated hips in plaster of Paris it used to be the custom to cover the pelvis and the region of the hip-joint almost completely with the plaster. The joint was covered so completely that it was sometimes impossible to get a satisfactory X-ray picture of it. Not only that, but they covered the joint so completely that access to it was not easy for the examining fingers. Likewise, the third thing that they did was to leave the plaster of Paris on for a greater or less length of time, usually

three months, and sometimes even longer, without removing it. The consequence was that not infrequently it was found, on removal of the plaster, that the hip that had been carefully replaced and put up in plaster of Paris was found to be out of its socket when the plaster was taken off; and one could not know when that change had occurred. When this occurred, the difficulty of treating the case was greatly increased, because the head had formed the habit of resting in another spot. It had made a new bed for itself to rest in—a spot other than that in which it should lie. This occurred to me, as it did to others, until I finally became so disgusted with the method that instead of doing the hips up in that way I made it a point to make the upper portion of the plaster cast quite thick, and then to cut away the plaster

FIG. 2



Method of applying plaster to allow of access to the head of the femur for purposes of examination and X-rays.

in front and behind so as to expose not only to the sense of touch, but likewise to view, the spot occupied by the head of the bone (Fig. 2). My finger is on the artery now, and the head is right under my finger. The result of this method of making the plaster cast was that when I would pass around the wards, if I was in doubt about a case, all I had to do was to slide my hand under the trochanter and push it up, and I could feel the head rise and fall with my finger in front. If I wanted to be still more positive, all I had to do was to have an X-ray made, and when I did, there was sufficient room, by slanting the tube slightly, for the rays to go down obliquely through the part without being obstructed by the plaster of Paris. That enabled me to keep the case absolutely under my observation, and it was easy for me to make examinations of the child from time to time to assure myself that the head was in proper position. Unless you do that,

you are apt to find, at the end of a certain length of time, when you decide to change the bandage, that all the time that you have been waiting for the head to fix itself in proper position it has been out of position. It may have gotten out immediately after being replaced, before the child was sent to bed, or it may have come out later while the child was in bed. In either case all the work had to be done over again.

TUBERCULOSIS OF THE HIP

This patient is a boy, thirteen years of age. There is nothing especially unusual about him from a pathologic standpoint. Here is the X-ray. It is a tuberculous left hip. You can see the tuberculous condition, involving all the upper portion of the head and neck of the femur and the acetabulum likewise. That was one of the earlier X-rays, and this one was taken about four weeks ago. Note the bending of the neck of the bone, but you see the diseased parts all the way in here. He had quite profuse suppuration. You can see the scars—the large scar anteriorly and three openings posteriorly. I might state that his is the only case of frank suppuration that we have in the ward of thirty-five beds. I remember the day very well when the tuberculous bone and joint cases in the wards of the hospital were profusely suppurative; and I think that even yet they have about as bad a name as has any affection. I mean that when you get suppuration of a tuberculous joint it is usually considered to be a very nasty and intractable condition still; but our experience has been that it is not so bad as has been painted, and in spite of our having a lot of tuberculous cases in our wards, we do not have many suppurating ones. This boy has been in the wards quite a while, but he is not a decrepit, emaciated youngster; on the contrary, he is improving, he has an excellent color, and is in good general condition. The only thing that now remains is to close his one remaining sinus. In spite of the extensive disease, he has considerable motion. I can move his limb around, especially in abduction, and he has no pain. The active symptoms are absolutely gone. Up to a short time ago we used to treat these cases in this way: In the first place, if we could determine that there were any sequestra there, we would not hesitate to go down and remove them. At other times, if it was a condition of caries that was accessible, we would go down and curette;

but almost never would we go down and deliberately cut out a piece of bone from the region of the tuberculous joint. In other words, we did not depend on operative measures for the cure of the case, but rather on conservative local and general treatment.

The general treatment consists in fresh air and sunlight; and sunlight, as nearly as we can apply it, by the Rollier method. Rollier is a Swiss surgeon of Leycin. His method consists in exposing the affected part to the direct rays of the sun. That is what we depend on to influence the case from the standpoint of the constitution. Then, in the local management, in the first place, we try to keep the patients quiet by means of plaster of Paris or other apparatus, in bed or out of bed, but usually in bed. Finally, we make local applications; and, of these local applications, the one that I have been partial to is washing out the sinus with dilute solution of iodine, gradually increasing the strength of the tincture until finally we would inject the pure tincture into the sinus—a small quantity of it. Alternatively we would use a ten per cent. iodoform emulsion in glycerine. Likewise, I sometimes use an iodoform wax. For instance, if I curette a carious spot, leaving a cavity, I fill that up with bone wax. Mosetig-Moorhof was the originator of the use of bone wax. At first I used to follow his formula, which called for forty per cent. of iodoform mixed with oil of sesame and beeswax; but sometimes we would not have the oil of sesame, and I thought that forty per cent. of iodoform rather high. Therefore I took beeswax, thinned it with olive oil, and mixed it with ten per cent. of iodoform, making an ordinary wax. That wax we used to put in the bone to fill up the cavity. When the method of Beck was devised, of using a bismuth paste, I used that quite often. I gradually fell away, however, from the use of bismuth paste for filling large cavities, because large amounts of bismuth are likely to be dangerous and poison the child. Lately we have mostly employed iodoform and glycerine or tincture of iodine, and have had fairly good results. Quite recently a new antiseptic has arisen, which we are trying now, and which we will try in the case of this boy, whose discharge has almost ceased, and that is the *Dichloramin T* antiseptic. This is a compound. I do not know its full history, but those who are associated with it are, first of all, Dakin, who is possibly primarily responsible for it, and, working with him, Doctor Lewis, of the Phipps

Institute, of this city; Doctors Le Conte, Lee, and one or two others whose names I do not recall. They used this Dichloramin T dissolved in an oil menstruum. It was intended to supplant the chlorine solution devised by Dakin and used by Carrel in the Carrel method of treating wounds. In the watery solution the antiseptic is so unstable that it parts from the watery vehicle in which it is carried so readily that in a short time after its insertion into the wounds it loses its power. The consequence is that in order for the wound to be under the influence of the antiseptic for any length of time it has to be flushed every two hours. When, however, the antiseptic is mixed with an oil, the oil holds the antiseptic more firmly and gives it off more gradually. The consequence is that you do not have to disturb the wound so often. It is sufficient to dress it once a day. That is what we have been doing with this case; and we have another case in the wards that has a scab all over it—that is, an ulcer. When it comes to superficial ulcers, the antiseptic is simply sprayed on; but in the sinuses it is injected. We insert on a probe a small pledget of cotton moistened with the oil, or we insert a grooved director, place the nozzle of the atomizer in the groove, and squirt it along the groove. Another way is to simply squirt a little in with a syringe.

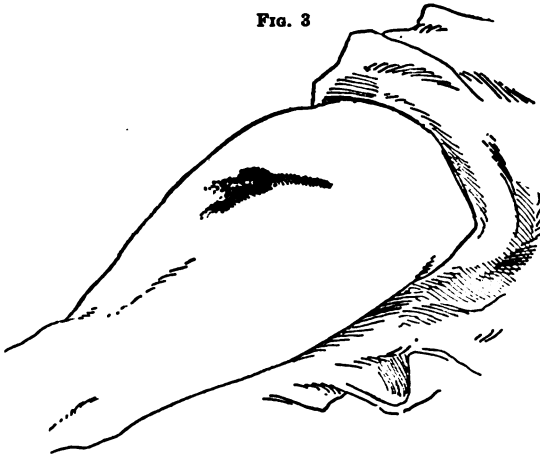
It has been used in this case, and the patient is getting well and the sinus is closing up. In some of the cases in which it has been used it is remarkable what results we get; and the antiseptic bids fair to demonstrate itself as the most efficient of all materials for the control of pus by local application to the suppurating area.

CHRONIC SINUS IN THE THIGH

It is very satisfactory to be able to solve everything definitely. For instance, a case comes up, and you know just what that case is and what has caused it. You know its pathology, and its treatment is evident. Here, however, is a case that came under our observation six years ago, and all the patient had was a sinus on the outer side of the thigh-bone below the trochanter. He is now seventeen years of age, and he was about eleven or twelve then. Now, why should a boy of that age get an abscess like that? When you think of lesions, collections of pus, etc., your mind goes to two things: either a tuberculous condition or a pyogenic condition. Is it pyogenic or is it tuberculous? We have never been able to tell which it was. You

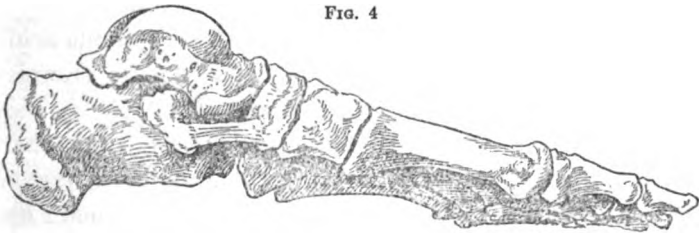
might say, "Why not try the tuberculin test?" Well, I have constitutional objections to using the tuberculin test by means of subcutaneous injection. The von Pirquet test can be used frequently, but it is not always reliable, nor of very much service when the patients begin to attain the age of this young man—fifteen to seventeen years. The next thing you do is to have an X-ray taken. We had X-ray plates made of his thigh-bone, and likewise of the sacro-iliac and

FIG. 3



Slightly discharging sinus of thigh.

FIG. 4



View of subastragalar joint from the inside. The long calcaneo-scapoid ligament is shown holding up the head of the astragalus.

pelvic regions. We had them made of all these, and absolutely nothing could be demonstrated by means of the X-ray to be the matter with either the femur or any of the pelvic bones, or even the spine. You see that it is near to the hip-joint, and yet on examining the hip-joint you see that he has perfectly free motion there in every way and that it is apparently absolutely healthy. He has been going around on the hip for six years, and it is still good. The sore healed

and then broke out once or twice again, but there is no evidence of any active disease in any of the bones of the pelvis nor of the thigh-bone.

You see he had once a big cavity there, and they injected bismuth paste three years ago. Later they took an X-ray of it. Here it is. You see the location? I suppose that the bismuth paste started from the opening there and, under pressure, migrated clear up above the crest of the ilium. Now comes the interpretation of the X-ray, and it has been suggested that at this point, around the neck of the femur, there is a suspicious-looking spot. The question is, Is that suspicious-looking spot disease of the bone? With that amount of bismuth paste strewn around, the shadow may be just as well of bismuth paste at that point as disease of the bone, and there is no positive evidence that there is any disease of the bone at all, in any way, shape or form. My experience has been that tuberculosis sometimes makes inroads in the soft as well as the hard parts. There occur practically tuberculous conditions affecting the skin, the fatty tissue under the skin, and the fibrous tissue under the fat, without disease of the bone. I have seen that, for instance, in the leg, and I have seen it up around the hip, buttock, and thigh.

In these cases, if they are mild, you can circumscribe them with an incision and cut right down through the fascia, cut them out, and sew the wound up, and the whole thing is done with. In other cases you can curette them out and wipe them out with chromic acid. We can also pack them with iodoform gauze, to stimulate granulations; and if you stir them up enough to increase their vitality they will heal up and granulate shut. This boy's case can be of that character. In this class of cases, however, if you treat these open, granulating wounds or ulcers with wet antiseptic dressings they remain open indefinitely—and this applies to other ulcers; for instance, take a *leg ulcer*—you can keep that in *statu quo*. The granulations become pale, the discharge ceases, and regeneration is at an absolute stand-still. Therefore, if I run across a case like that in the leg, I may be tempted to discard all antiseptic dressings and apply a large poultice of flaxseed, with some resin cerate spread on it. The poultice produces heat and moisture, and the resin serum adds the ginger to it. Pus comes pouring out, but the granulations wake up and begin to look like cobble-stones. They become round and big, and

shoot up all over the ulcerated area to the surface. Finally, when I get the granulations up high enough, I stop this treatment, for I have got the lesion out of its bad course. When you get a person out of his rut it is easy to manage him. It is the same with a leg ulcer. When you get the granulations shooting up well, you can cure the ulcer easily. The moral is that if you want these sluggish cases to heal you must stimulate them and put some life in them. If you do, you can cure them. The average recent graduate, whose actual hospital experience has been limited, when he encounters an ulcer of this sort, is apt to put on a plain gauze or antiseptic dressing. This will keep it clean, but the ulcer just stays quiet and does not do anything. That is not satisfactory. We must see things move in one way or the other. If they move in our direction, all is well, but if they do not, then we seek something else to make them do so.

CHRONIC SINUS IN THE THIGH

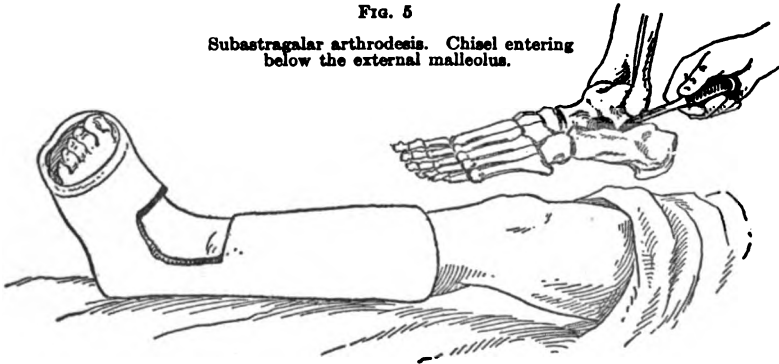
Here is another ulcer case, about three inches below the trochanter on the outer surface of the thigh. We tried the Dichloramin T on him. This patient has been probed. One of my assistants opened it to hunt for a bone lesion and could not find any, and the probability is that there is no serious bone lesion there. The ulcerated surface is now scabbed over and is rapidly healing.

I have no hesitancy in repeatedly showing you cases of the same affection. To illustrate: A person will have a familiar object (in this instance a skeleton foot) presented to him and will say, "I have seen that before." Very well. Then comes the question, "Have you seen everything about it that is to be seen?" When you say "everything," you mean a good bit; because I am sure that I can hand the object to one individual and ask him to describe it, and he will describe certain characteristics; and if I hand it to another, he will give certain characteristics that he observes, but his description will not tally with that of the first man. If I hand it to a third, he will observe something that the other two failed to see; and if I go on, almost every man will find something different in the object from what the others did. If that is so, then suppose I hand it to one gentleman, and he looks at it and describes it, and returns it to me, and then I hand it to him again; the second time he handles it he

may see the point that the second man brought out. The third time I give it to him he will pick out, perhaps, the thing that the third man had discovered; and so on. If I present the object ten times to him, instead of looking at the same thing each time, if he is using his eyes properly, he is discovering something new each time he looks at it. Therefore I have no compunction in presenting either a subject or an object to you again and again; and I expect you to learn something from it every time you see it, if you use your eyes as you should.

Take the bones, for instance. I am rather fond of the bones, because they are really very interesting. When I was your age I used to wander around with the carpal bones in my pocket. I got

FIG. 5
Subastragalar arthrodesis. Chisel entering below the external malleolus.



Cast out away in front of ankle to allow for swelling in club-foot cases.

so familiar with them that I could identify the individual ones by feeling their different shapes. Wolff enunciated the principle that the shape of a bone is dependent on the stress to which it has been subjected. In other words, if you desire a bone to grow right, it must be put under normal conditions. If it is put under abnormal conditions, it grows in a different way. In knock-knee, for instance, if the femur has a pressure that is greater on the outer condyle than on the inner condyle, the pressure on the outer condyle keeps it from growing as rapidly as the inner condyle. In a knock-knee, if the leg is in extension, the internal condyle is longer than the external condyle and the leg tilts outward. When you bend it to a right angle you find that the leg comes up in line with the thigh and does not show the deviation it does when the knee is extended. This is because the posterior surfaces of the condyles are alike and therefore

they grow evenly and not unevenly, as does the inferior surface. To such an extent is this so that it was proposed to treat a knock-knee with the legs in a bent position, to give the external condyle a chance to grow out; but, needless to say, that method of treatment was never put to any extensive use.

Now let us return to the foot again. The foot articulates with the leg and by a hinge-joint. Therefore the movements of the ankle-joint are of a hinge nature—anteroposterior. Then, in addition to that anteroposterior movement, a part of the remaining portion of the foot, the portion beyond the ankle-joint, has a movement, so to speak, between the individual bones. For instance, you can twist your foot in like that and twist it out like that. This is evidently intended so that when you put your foot on an unequal surface it will rotate sideways in and rotate out. That movement can not occur at the ankle-joint, because the ankle-joint is a hinge-joint. There is a little rotary motion in the ankle-joint when the joint is extended, but it is comparatively slight, and the motion in the ankle is practically only anteroposterior. If that is so, where does this lateral motion of the foot take place?

It takes place in this joint, which I have called, for convenience's sake, the subastragalar joint. The motion occurs in the joint beneath the astragalus. It rocks sidewise. That joint is formed by the calcaneum and the astragalus posteriorly, then the long calcaneoscaphoid ligament and then the astragaloscaphoid joint anteriorly; the cuboid bone does not enter into it. The foot rocks laterally on the astragalus.

If you look in your anatomy for the subastragalar joint that I have described, you will not find it, because they have classified their joints, not according to function, but according to structure. You will find a description of the calcaneo-astragaloid joint and one of the astragaloscaphoid joint, but you will not find any of the subastragalar joint. You will, however, find me using the term subastragalar joint quite frequently, and I trust that you will understand me.

PES VALGUS DUE TO INFANTILE PARALYSIS

We have here a case of pes valgus from infantile paralysis that we shall operate on by subastragalar arthrodesis. Arthrodesis means making an artificial ankylosis. We wish to eliminate the rocking

motion permitted at the subastragalar joint, and desire the astragalus to become attached to the bones with which it comes in contact, viz., the scaphoid in front and the calcaneum behind and below. It does not come in contact with the cuboid nor with the cuneiform bones.

We make two small lateral incisions: the one on the outer side is opposite the tip of the external malleolus; the other, about an inch long, is about three-fourths of an inch below and in front of the internal malleolus. The posterior portion of the astragalocalcaneal joint is reached through the outer incision, and the adjacent surfaces of the scaphoid and astragalus and sustentaculum tali and astragalus through the inner incision.

The way we do an arthrodesis in this clinic is with a great deal of brutality. The procedure consists not in paring off the adjacent cartilaginous surfaces, but in taking a small quarter-of-an-inch-wide gouge and thrusting it between the bones, churning it around, digging up the line of the joint right across below the external malleolus (Fig. 5). You push the gouge in and dig the cartilage up, and make the parts as rough and disturbed as possible. That being done, withdraw the gouge and incline it anteriorly, and dig up the outer portion of the head of the astragalus and the adjoining articular surface of the scaphoid. On the inner side the landmark is the internal malleolus. Three-fourths of an inch below its tip is the sustentaculum tali. You know the subastragalar joint is between the sustentaculum tali below and the astragalus above, so you go three-fourths of an inch below the internal malleolus; but you do not jab in the same way as was done on the outer side, because if you do, you are liable to cut the posterior tibial tendon.

This tendon, which comes from behind the internal malleolus, passes downward and forward to be attached to the tubercle of the scaphoid. It would not be a very dreadful thing if it were cut accidentally, but there is no use in doing careless work. We make an incision, hook the tendon out of the way, and push the chisel in and dig the surface of the joint, and then go in between the astragalus and the scaphoid, and churn and dig all these surfaces up, so that we have a mass of jumbled-up pieces of bone and cartilage. We leave them that way. We do not take any parts out at all. As a rule, we get very good union.

ARTHRODESIS

In this case the operation of arthrodesis was done for a valgoid condition. The feet were everted, and we put them in a slightly over-corrected position. As you see, they have a little tendency to varus, which is intentional. It was done six or seven weeks ago. Grasping the foot with the fingers of the left hand just below and in front of the malleoli to fix the astragalus, with the other hand we try to move the os calcis, but it does not move; the two bones are solidifying into one piece, but of sufficient thickness and immobility to indicate that even though the patient is but seven years of age enough inflammatory matter has been poured out to cause a complete ankylosis of the astragalus with the surrounding bones. It is now a fibrocartilaginous mass, which, at the end of seven weeks after the operation, has already assumed a considerable amount of solidity. The operation was a subastragalar arthrodesis. We have not interfered with the ankle movements at all.

I should like you to understand and appreciate this operation of arthrodesis, which has as its object the stabilizing a foot. Weak feet, flail feet, and other deformed feet are proper subjects for it. I am convinced that the operation of arthrodesis, or the making of an artificial ankylosis, is, in the first place, a great utilitarian procedure. In my experience, it has been one of the most useful procedures for the treatment of weak, flail, and deformed feet that we have. In the next place, I am convinced that it is not extensively enough practised. It is not used so much as it should be.

One reason that arthrodesis is not popular is because at first it was thought to be necessary to turn the joint inside out and pare off the cartilage, so as to bring the pared surfaces of the bones together, where they must be held until union takes place. One man happened to succeed in doing this, but, so far as I have heard, few have succeeded in making arthrodesis satisfactorily according to that formula. Others who tried it did not get the immobility that they had tried for. That occurred so often that the operation became discredited. It occurred to me, as well as to others, when I tried it; so I afterwards took an entirely different course. Instead of trying to be as careful as I could, I tried to be as rough as possible. I dug up the joint and left it in as rough a condition as I could, with the idea that inflammation would ensue and unite the separate bony

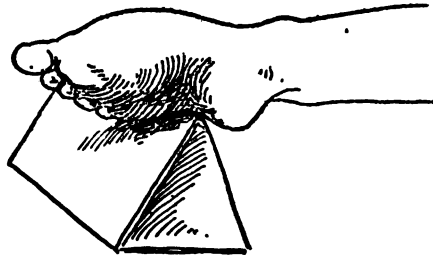
and cartilaginous particles in one mass, making a firm union. That is the method I have followed for several years in all our arthrodeses, and you can see that even in this child of seven years of age, at the end of six or seven weeks, union is so firm that you would find it rather hard, if you wanted to put the foot in a valgus position, to do so again, for you would find it difficult to push it out. It is thick and boggy there already, showing that the inflammation has already soldered things firmly together.

CLUB-FOOT

The next case is stretching of a club-foot. There are two kinds of affections of the feet. One is a weak foot, and the other is a strong foot. The strong foot you fight, and the weak one you nurse and coddle. Flat-foot is the typical example of the weak foot, and club-foot is the typical example of the strong foot.

We have learned much of recent years in regard to the extent which the foot will stand what may be termed mauling, or—to

FIG. 6



Stretching a club-foot over Koenig's block.

use a scientific term—manipulation. A club-foot is corrected by twisting it outward. If the patient is a very young child and the deformity is not very marked, you can succeed in correcting it with your hands by manual stretching. If, however, the hands are not sufficient, you may employ a block devised by Professor König, formerly of Göttingen, afterward of Berlin, and now dead. It is a wedge-shaped block of wood, slightly padded on its edge. You put the foot over it and bend it down (Fig. 6).

This is Thomas's wrench, invented by the late Mr. Thomas, of Liverpool, and used by Col. Sir Robert Jones and others (Fig. 7).

This is the Rizzoli osteoclast (Fig. 8). It is primarily intended for the long bones, but can be used for obstinate cases of club-foot.

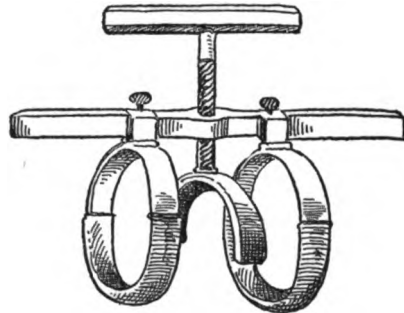
These are two instruments that we have constructed for our own private ends, and we also use them for stretching the foot. It will not do to simply cut the tendons and then put the foot up in plaster

FIG. 7



Thomas's wrench.

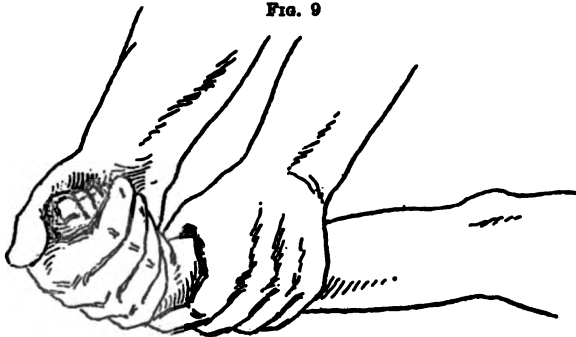
FIG. 8



Osteoclast of Rizzoli.

and let it go at that. This is a pretty bad club-foot. Never cut the tendo Achillis at the beginning of an operative procedure, because you need it in order to form a point of counter-pressure. In stretching a club-foot, I grasp the leg down well toward the ankle, and then take the foot in my other hand (I use the right hand for the right

FIG. 9

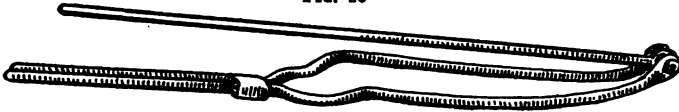


Stretching a club-foot by hand.

foot and the left hand for the left foot) and rotate the anterior part of the foot outward. Holding it out like that, I bend it up (Fig. 9). By bending the knee, I relax the tendo Achillis to a considerable extent and get it approximately into shape. But I can not get it as well as I want it, so I take König's block.

I put the child on its face and lay the foot over the block, like that. I hold the heel down with one hand and the fore part of the foot with the other and bring the weight of my body to bear on it. Now it has gotten down pretty well, but hardly enough yet. You can appreciate how much strength is required to do it. However, I

FIG. 10

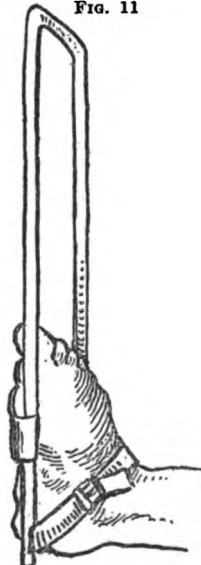


Osteoclast to use on bow-legs, club-feet, etc.

can return to the attack and resort to this, which is, as you see, a U-shaped bar (Fig. 10).

I take a towel, place it around the foot, and place the instrument over it. Then I put the upper end under my axilla and place

FIG. 11



Club-foot stretcher.

my hands so, and, by a rocking motion, I can twist the foot and stretch the tendo Achillis still more. Now I bring it down, and the foot comes out pretty well. I can overcorrect it (Fig. 11).

The stronger, bigger, and heavier you are, the better you can do

this. There, I think that is enough. Where there was formerly a hump on the instep there is now a hollow. I will now leave this and put it up in plaster of Paris, so. We have not divided the tendo Achillis, as it looks as if it had been done on a previous occasion.

There is one other thing that I wish to speak of. Sometimes I am still not satisfied with the result, so I then resort to an appliance which is perfectly simple in its mechanism. We rest the sole of the foot on the two bars below and then gently, but firmly, cause the lever to descend and the projecting hump is pushed down to the extent we desire. This can be done without breaking the skin, the foot being somewhat protected by wrapping a towel around it.

You would think that there would be a dreadful reaction from this procedure, but we do not find it so. We do not have trouble from overmanipulations, but we may have trouble due to the pressure of the plaster applied in our endeavor to hold the foot in its corrected position.

I will not stretch either of these feet any more. They come out straight enough. I believe in moderation, although you might not think so from this demonstration; but moderation is relative. If I found that after such a stretching as I did on this foot there was a great reaction around the top of the foot or that there was a widespread ecchymosis and dreadful swelling, with possible gangrenous spots, why, then, I should feel that I had been too rough and had gone to an unwise length; but, as I have said, the only trouble that we have with these club-feet is that sometimes, in our anxiety to make a good correction, we put them up with a little too much pressure on the sole of the foot. If you do this, a slough may form at the outer edge, over the region of the cuboid. That, however, is not due to the primary manipulation, but to the pressure of the cast. To guard against it we cut the cast down the front to allow for swelling and look at the foot every day or so, or possibly take the cast off in a few days and apply a new one.

I give an anodyne to the child afterward, because there is no necessity to allow it to suffer after these procedures. I keep the foot elevated, and by the next day the tension has to a certain extent subsided. The temperature does not go up to any extent, and the child is soon comfortable and does not mind it.

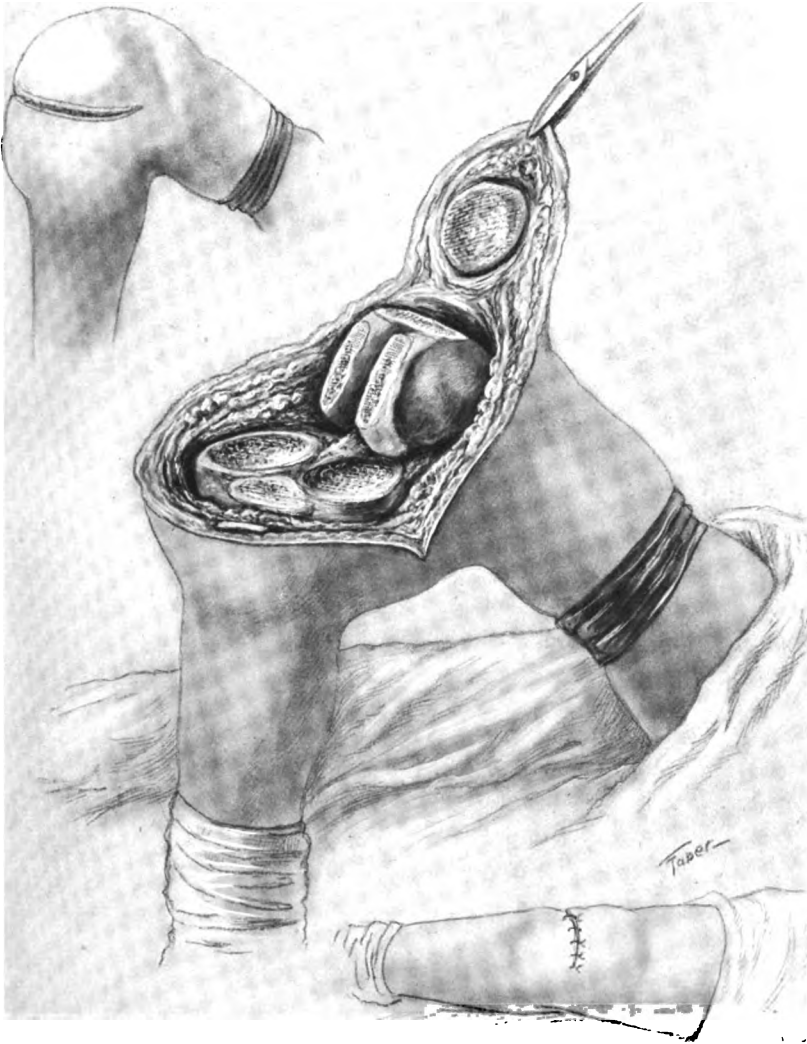
TUBERCULOSIS OF THE KNEE-JOINT

I have one more case that I want to show you, and that is a tuberculous knee in an adult—a young man.

Now in this case I am going to do a *resection of the knee*, but hardly a strictly orthodox resection. A few years ago resection of the knee used to be a very popular operation, and a prominent exponent of it was Doctor Phelps, of New York. He used to do it in nine minutes, and he could do it and put the plaster and everything else on in thirteen minutes. He worked pretty fast in order to do that. While such things can be done, it is not necessary to do them in that way. In doing these operations, you must remember that you start with a definite object in view. The bulk of the orthopædic patients are in more or less good general condition. The operations are not emergency procedures. There is, as a rule, no need for haste, and these operations demand exactness. Take, for instance, the operation of resection of the knee-joint. This is done with the object of removing a certain amount of diseased tissue and ankylosing the knee-joint, thus causing any disease that exists there to be subdued; to remove the signs and symptoms, and put the limb in such a condition that it will remain serviceable. If you are not careful, you will fail in accomplishing these objects. In other words, in all orthopædic operations you will find that if you do not do them exactly right in the first place, and attend to them properly in the second place, that in the third place you will be required to do them over again. Every now and then you will see me doing something over again, which means that I did not succeed in accomplishing my entire object at the first trial. How much this was due to the inherent difficulties of the case, how much to inordinate ambition on my part, and how much to downright ignorance or lack of ability you will have to decide for yourselves. You can be sure, however, that if you do not do everything just right there is no use in your doing it at all. Therefore, as a rule, I do not attempt to be rapid, but I do attempt to be exact in my operative procedure.

The reason we are going to operate in this case is, first, because the joint is distinctly diseased. I do not think that the bone is very markedly affected, but it is a very clear and typical case of tuberculosis of the joint, and it is in a young adult. If I were to treat him conservatively, the best that I could hope to do for him, after a long

FIG. 12



Resection of knee, showing exposed bone surfaces to obtain fixation.

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period of treatment, would be to subdue the active symptoms. The knee-joint might get quiet, but the knee would remain fixed, we will say, in a straight position; but it would not be firmly fixed. It would not be ankylosed. It would only be fixed by the contraction of the soft parts; and, ten to one, in going around with a practically stiff knee from this disorganized joint, he would sprain it. I mean by this that he would bend it, rupture some of the ligaments, and have a flare-up again. He would then be disabled and in bed for quite a time. After that possibly it would all quiet down again, and he would get up; but he would probably proceed to injure the knee again, and it would flare up once more. This time it might not quiet down so readily, and might start to suppurate. In other words, it would not be a serviceable, dependable, or reliable knee. So, even if I treated it conservatively, in spite of the expenditure of much time and labor, I should have at the end an unreliable knee.

In a person of this patient's age, in whom growth has ceased, I think that it is justifiable to resect a sufficient amount of the articular surfaces to get a bony ankylosis and make a stable union, right square across. I should not need to remove a very large amount of bone in order to do that. All that I have to do is to make a good exposure and see that the cartilage is removed over the bone, so that I have a good apposition of bone to bone. I shall try to be careful and remove only just enough bone in front to allow me to bring the leg straight without dividing the posterior ligament. In other words, I shall try to remove just enough so that when I put pressure on the two surfaces will come tightly together and there will be no necessity for using any foreign bodies to fix the bones and keep them from moving.

A considerable amount of credit is due Dr. Leonard Ely for advocating the view that in order for tuberculosis to cease its activity in a joint it is simply necessary to fix the joint. That was his preaching; but still others had an inkling of the same truth, because Lorenz evolved a plan of treating hip-joint disease (coxalgia) by means of the short spica and allowing the patient to walk around on the limb. This favored an ankylosis of the hip-joint; and that is what he was aiming for. He claimed that if he got a good ankylosis of the hip-joint it was better for the patient than to have it cured by conservative means and have motion. He may have been right in

that; but Americans, as a rule, have not accepted his principle of allowing tuberculous children to walk around on the hip-joint—although there are some that follow him.

John Ridlon, of Chicago, claims that tuberculosis of the joints in adults can be cured by the same methods as tuberculosis of the joints in children, and I think that is, to a large extent, true. I have no doubt that if we persisted in treating this case conservatively we could get all the symptoms to subside and most of the signs to disappear, but I do not think it would be worth while. Although it is possible, I do not believe that it is best for the man, because, even if we should succeed in subduing everything, we should still have an unreliable knee.

Now look at the knee before it is disfigured by the iodine. That is a typical tuberculous knee. It is white in color, swollen, and boggy to the touch. You can not tell the exact amount of involvement in the knee. The cartilage may be gone; there may be suppuration and pus, and certainly the synovial fringes are markedly involved.

I do not believe in using the Esmarch bandage—that is, the wide rubber bandage. We never use that. But we will use the Esmarch tourniquet. It is not necessary, in ordinary resection, to put on an Esmarch tourniquet, but it is simply a little convenience.

It is easier for me to cut from the right side, because I am right-handed, than to cut backward from the left side. I will make the incision from one side to the other, right across, below the patella. I will cut the ligamentum patella and go directly into the knee-joint, right square across, and then put my finger below the patella, dissect it up, and flex the knee. See the pus well out. It is not actual pus, but synovial fluid and detritus. Then I will prolong the incision on each side and be rather careful about cutting the crucial ligaments, because I do not want to injure the artery which lies close behind them.

I forgot to say that we have tapped this knee-joint and had the fluid examined, and tubercle bacilli were recovered from it. Here are the crucial ligaments. I cut them away. Now I shall take the gouge and gouge the top surface of the tibia away. I turn up the patella and clean out the subfemoral bursa.

This is the surface of the condyle, and I am clearing off the

tuberculous synovial tissue and now saw off just sufficient to allow the knee to come straight. Now you see the under surface of the patella how it is eroded. I will take a saw and slice the under surface off. Now I have got a clean, fresh surface of the under surface of the patella to unite with the upper surface of the femur and tibia.

I like to put a drainage-tube in, and we will take it out in twenty-four, thirty-six, or forty-eight hours. I do not often leave it there longer than forty-eight hours. I am afraid of hemorrhage or abundant oozing, which, if I did not allow to escape, would distend the wound and cause suppuration.

We will first fix the leg on a long, straight splint and then give additional fixation with plaster-of-Paris bandages. I am rather fond of this combined way of using a splint and plaster. I take care not to have the heel on the splint, but allow it to project beyond so as to avoid pressure. We elevate the leg and the next day take a pair of scissors and cut a hole in the bandage, remove the cotton, and pull the drainage-tube out, but we will not disturb the dressings at all.

BOW-LEGS

We have here a bad case of bow-legs. This child is only two years and a half old, which is rather young to operate on. In some circumstances I would not do it; but look at those legs! Here operation seems justified.

We put the leg on König's block and forcibly straighten it out. I think I have produced a green-stick fracture. See how nice and straight it comes. It will not stay right, though. We will first bandage the legs to wooden splints and then put them up in plaster. I will put cotton around the legs in the present case. I think cotton is all right in cases in which you have exercised considerable trauma, but in a good many cases you do not need to use anything except a simple bandage. I like to carry the bandage up around the hips and keep the foot at right angles to the leg.

Using an osteoclast as I did to break the second tibia may seem like primitive surgery; but if there is any better way to do it, I want to know it. That is about the only thing I care to use an osteoclast for. I prefer it to osteotomy for young children.

Codivilla, in correcting knock-knee, used to do what he called an epiphyseolysis, in which he detached the epiphysis of the femur

from the shaft of the femur. He broke the epiphysis off from the diaphysis, and so corrected the knock-knee. There are a good many who would not do that. I would not, for instance; and there are a good many others like me. Codivilla was asked if he did not interfere with the growth of the limb in breaking through the epiphyseal line. "No," he replied, "I do not." To prove his point, he followed up a lot of his cases that had had this detachment of the epiphysis, and in none did he find that there had been any interference with the growth of the bone caused by it. Therefore, if I should accidentally get detachment of the epiphysis, as has happened to me on some occasions, I would feel that it was not such a serious thing.

**COMPOUND COMMINUTED FRACTURE OF THE TIBIA
AND FIBULA FROM RAILROAD INJURY; GENERAL
CONSIDERATIONS OF ABDOMINAL SURGERY; IM-
PORTANCE OF DIAGNOSIS AND THERAPY; FASCIAL
SARCOMA IN LOWER QUADRANT OF ABDOMEN;
COLEY'S TOXINS AND THE USE OF THE X-RAY IN
THE TREATMENT OF SARCOMA; SARCOMA OF THE
ILIUM; REMARKS ON X-RAY BURNS; INJURY OF
THE PELVIS, SEPARATION AT THE SYMPHYSIS, EX-
TERNAL URETHROTOMY, AND SUPRAPUBIC DRAIN-
AGE OF THE BLADDER**

A SURGICAL CLINIC

BY ARTHUR DEAN BEVAN, M.D.

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COMPOUND COMMINUTED FRACTURE OF THE TIBIA AND FIBULA

I WANT to call your attention to this patient, whose case illustrates in a certain way some of the problems that are met with in war surgery.

About sixteen months ago this man had a severe smash of the right leg. He was in a railroad wreck, and either a wheel went over the leg or struck it and shoved it off from the rail, injuring it badly. One of these two things happened. It was an empty box car which weighs somewhere near twenty tons. That opens up a rather interesting problem. I may not have time to speak of this at any length this morning, but usually when a limb is run over by a car wheel and is crushed between the rail and the wheel all of the tissues are so devitalized that amputation is necessary. Not infrequently, as this patient expressed it, the limb is caught and mashed, but not actually run over. It is shoved along between the rail and the wheel and the force produces a bad smash, but does not devitalize the entire limb. At any rate, he sustained a severe compound fracture of both bones, and it is not only compound but comminuted. A large fragment of the tibia was separated from the shaft above and from the shaft below, and, in addition, there was a fracture of the fibula.

I think he was operated on once before I saw him. It was well handled for a considerable period, but resulted in nonunion.

We operated on him on the 14th of June, about four months ago, and had great difficulty in making a good piece of carpentry work, because the bones were so out of shape that it was difficult for us to bring them in good position; but finally, after a good deal of effort, and dividing both the tibia and fibula, we put in a steel plate, with four screws in the tibia, and brought the fibula into very good position. It healed up quite well. There was some slight suppuration, but the steel plate has healed in; we have not taken it out. We put on a cast above the knee when I saw him, and took this cast off about six weeks ago, although there was not as yet complete union. As I took hold of the foot I could find motion still at the point of fracture. Of course, this was in an infected area from the old compound comminuted fracture, and the process of repair was not very rapid, and yet it has been more than ordinarily good in this case. The man's general condition is good.

To-day he comes back here, and I find that there is good, stiff union of the fragments. The limb is very different in appearance from the other limb because he has not walked on it for a year and four months. He has what is known as plaster-of-Paris disease by some surgeons. That is simply a catchword, but it means this: that if a man has a limb put up in a plaster-of-Paris cast for some months, or does not use a limb like this for a period of a year or more, there is very marked atrophy in the bones, so that if you were to take an X-ray picture of this leg that has not been used for a year and compare it with the leg that has been used right along you will find the bone in the good leg is much denser and will throw a much more definite shadow in the X-ray, while the bones of the injured limb look as though they had been absorbed and rarefied. This is entirely due to what is technically called the atrophy of disuse. The muscles in this leg are not nearly so good as the muscles of the other leg, and that is due to the atrophy of disuse, and exactly the same thing happens in a bone; and you can see a difference in the shadow cast by the X-ray in a limb that is functioning all the time and in a leg that is not being used.

This man has a condition which demands very good, commonsense after-management. He has a swollen knee-joint, and that swollen knee-joint is very painful if he uses it too much. His ankle-joint is

quite stiff. The muscles of this limb are not nearly so strong as they were before he was hurt, and the bone is not so dense. The problem is to obtain the best possible functional results, and that is a simple one, now that we have obtained union, if it is carried out the right way. For several weeks he should walk on this limb with crutches. He needs to do more than this. While he is in bed he should bend the knee-joint, and gradually he will get a return of function. In the next place, he should sit in a chair and practise moving the leg up and down at the knee and in the ankle. At first, in performing these exercises the limb will swell and be painful, but he has got to grin and bear that for a little while, and if the swelling becomes too great he can for a little while wear a flannel bandage that extends from the toes up to above the knee. The whole thing is to begin to use this limb gradually, in a commonsense way.

He should walk on crutches, and at first he should put a little weight on the foot, and then gradually he should increase that weight as the limb becomes stronger. In two or three weeks he will throw away one crutch and use a crutch and a cane; then in about two weeks more he will throw away the other crutch and then use a cane. He ought to be cautioned not to slip and break this leg over again. Any frosty morning he might want to go out he should put on a pair of rubbers. He must be exceedingly careful not to slip and hurt this limb. It will take two or three months before he will be able to walk well. Gradually the muscles will become stronger and the bone denser. Inside of a year he will probably be able to walk five miles, and the end-result will be something like this: there will be shortening of the leg, but this will not be a matter of very great moment, because he can have an insole in the shoe and have the heel made a little higher. He should not do that at first; not until he gets to walking well. If we do not obtain union in a case of this kind, the man is incapacitated for the rest of his life. If he does get union, he will be restored to 90 per cent. of his former efficiency.

GENERAL CONSIDERATION OF ABDOMINAL SURGERY

This morning I want to take up some general considerations in regard to abdominal surgery. I want to discuss this problem from a scientific standpoint. I want you to have this conception of clinical surgery with special reference to abdominal surgery: that each case

that comes to you is to be studied in a scientific way and is a case of clinical research. It should be looked upon as a scientific research of a clinical case.

You students have all had a very good training in biology, chemistry, physics, and in your laboratory studies in medicine, in anatomy, pathology, physiology, and pharmacology. Now you come to a point where you must apply these sciences to a concrete clinical case or to a group of cases. You want to learn that the modern scientific surgeon looks upon a case such as I shall show you this morning as a scientific problem, and he handles it in exactly the same way as a chemist does his problems in the laboratory when a substance is submitted to him for qualitative and quantitative analysis. It is in every sense just as truly scientific as is the problem of the chemist.

We find, in our analysis of these cases, that there are some things that we do not know. We find very often that there is insufficient evidence; that after the most exhaustive study of the case it is impossible for us to obtain sufficient evidence upon which to base an absolute, scientific, and final diagnosis. Very often a final diagnosis is not arrived at until the case comes to an exploratory operation or to the post-mortem room. Nevertheless, the case should be handled from the purely scientific standpoint of obtaining all of the evidence that can be gathered, of analyzing that evidence and of interpreting it properly so that we can make a correct diagnosis.

Then the second function of the modern surgeon comes into play, and that is the function of therapy. In a clinical problem, therefore, there are two great considerations to be kept in mind—the science of diagnosis and the science of therapy.

In this particular case that we have this morning we find a man, about thirty-five or forty years of age, who comes to us with a large mass in the right lower quadrant of the abdomen. This mass is as large as a child's head. It is in the abdominal cavity. It has grown to this size within four months. We have been able to obtain first the following piece of evidence: About four months ago he had an acute abdominal attack of pain that passed away within a few days. Following that, within a few weeks, he discovered gradually the development of this large mass in the abdomen. We have attempted to apply to this case the methods of diagnosis which are known in such cases. We have taken this man's temperature, and we find that it

is not elevated. We have had the blood examined, but it has given us no definite information. We also have had his urine examined, with negative results. We have taken a very exhaustive clinical history, and nothing has been learned from this that determines either the definite anatomical location or a definite pathology of the lesion. We have examined his fæces, and there is no blood in them. We have taken an X-ray picture, but have learned nothing as to what this mass is. We have fluoroscoped the patient, and after filling the colon with barium solution there is nothing in that fluoroscopic examination which enables us to determine what this tumor or mass is, either anatomically or pathologically. Analyzing the clinical history, we can not find enough evidence on which to base a diagnosis. Analyzing the evidence that we have obtained by these examinations, we are unable to find enough evidence on which to base a definite diagnosis. The physical examination gives us probably more evidence in this case than anything else. As I have said, there is a large, hard mass in the right lower quadrant of the abdomen. It does not fluctuate. It does not seem to be cystic. It is hard, firm, and rather regular. It is fixed and not movable. There are some things that we can say, from the evidence that we have obtained, with some degree of probability, if not certainty, that there are no obstructive symptoms of the bowel. There is no blood in the fæces, and from these two facts it is probably not a tumor of the bowel. I would not say that with absolute assurance, but simply as a matter of probability. Examination of the urine and examination of the tumor speak against its being a tumor of the kidney. There is no blood in the urine, but that is only a probable diagnosis, because not infrequently we find huge tumors of the kidney, like hypernephroma, in which there is no blood in the urine. Blood occurs in the urine in about 50 per cent. of our cases of hypernephroma.

Because I do not want to keep this patient unnecessarily long under the anæsthetic, we will proceed to a further means of diagnosis—an exploratory operation. Am I willing to venture an opinion in this case? From the clinical evidence, I shall venture this clinical diagnosis as most probable; namely, a fascial sarcoma. Shall I exclude definitely other conditions? Not with any degree of certainty. I cut down through a muscle-splitting incision and find a huge neoplasm, and as I go through the skin and divide the superficial

fascia and other structures and separate the peritoneum I come upon this tumor. It is not an abscess. It is almost certainly a fascial sarcoma. The X-ray showed no picture of bone. There was nothing in the kidney; there was nothing in the bowel that pointed with any great probability to a lesion either of the bowel or of the kidney.

What shall we do with it? This tumor is as big as this man's head. It fills up this entire space. Would it do any good to remove it? Not at all. From the standpoint of a radical operation, we can say that the conditions are such that a radical operation would give no prospect of benefit. Anatomically this tumor is so situated that we could not remove it and remove at the same time all of the malignant cells of that tumor. It would be inconsistent with the life of the individual. Therefore, what we have done here this morning is to make an exploratory operation and remove a section of this tissue for microscopic examination. Granted that we find that this is a sarcoma—and I will make a report of this case to you at the next clinic—what are we going to do with the case? Shall we tell the man to-morrow morning or in a day or two that he has a cancer—using the term cancer in the broad sense of covering malignant neoplasms—and that it is inoperable and hopeless? Not at all. As physicians you have a real duty to perform in such a case as this. You should do what you can to make this man physically and mentally comfortable and do what you can to prolong his life and inhibit the growth of the tumor.

Have we any agencies that are of value in doing that in this particular case? While it is rather doubtful, it is worth trying, and there are two agencies that can be employed and which I shall use in this case with the idea possibly of inhibiting the growth of the tumor and possibly of bringing about its disappearance. One is the X-ray and the other is the use of the toxins of erysipelas. Of these two I have very much more confidence in the use of the X-ray in this particular group of cases than I would in the toxins of erysipelas. In the management of this case I want you to be impressed with the fact that I regard it as a scientific management from the standpoint of our present knowledge; namely, to use the X-ray with the idea of producing a diminution in, or disappearance of, this mass. In advising that, however, you should be careful to advise that it be given by an expert, because there are real and even great dangers

in the therapeutic use of the X-ray. We have seen some very unfortunate cases where a patient of this kind will come to us very badly burned by the X-ray—burns which produce a great amount of suffering.

In the use of the X-ray in a case of this kind it should be employed by an expert and shorn of any risk of producing serious burns. The argument for that is that the use of the X-ray in a case of this kind is an uncertain thing. It simply holds out a possible means of benefit, and we are not warranted in pushing it to the point where we will injure the patient with the hope that it may produce improvement or cure. There are some very remarkable and exceptional results from the use of the X-ray in some of these cases. In some instances the tumor diminishes in size as a leukæmic spleen diminishes under X-ray treatment. Some of them diminish in size as masses of Hodgkin's disease diminish temporarily under the X-ray treatment, and during that period, which may cover three months or even a year, the patient is hopeful. I have had patients who were so greatly improved from the use of the X-ray that they were able to return to their work, and they felt they were cured of the condition, and this has happened especially in some of these sarcomas in the abdomen. Therefore we should give the X-ray a very thorough trial in this case.

The second agent that may be tried is that of the toxins of erysipelas. I want to say a word or two about this phase of the subject from a chronological standpoint. When I was a student in this clinic Doctor Gunn, who was our professor of surgery, used to tell us he was very much impressed with the fact that some of his cases of malignant disease which were operated upon and then developed in the course of after-treatment an attack of erysipelas were cases in which he was most apt to obtain permanent cures. That legend was quite common among the surgeons of 1860, 1870, and 1880, and many surgeons of wide experience were able to refer to cases in their experience where a malignant tumor had diminished greatly in size after an accidental attack of erysipelas, or an attack of erysipelas complicating the operation for the removal of a malignant tumor, so that it was not at all surprising that when Fehleisen discovered the streptococcus of erysipelas the first thing he did with the pure cultures he obtained was to treat a group of malignant tumors with the hope that by producing an artificial erysipelas with these

pure cultures it might lead to cures in these cancer cases. Some of his results were rather encouraging, but the treatment was too fatal. Too many of the patients died from the attack of erysipelas. So a step further was taken, and that was to take a culture of erysipelas, kill the germs and use the toxins, injecting them with the hope that they would produce a diminution in the size of the growth. I think most of you in this senior class are familiar with this work under the name of Coley's toxins or Coley's fluid, as it is known in the literature. Coley added to the pure culture of streptococcus of erysipelas, prodigiosus, with the idea that it would strengthen the effect of the toxin of erysipelas, and the Coley fluid is a mixture in pure culture of erysipelas and of prodigiosus. Coley himself has done a remarkable and enormous amount of work in the last twenty-five years with this agent and has claimed very excellent results. No one else has obtained with this treatment such good results as Coley.

The general impression of the profession has been that it is not a cancer cure, nor a cure for a limited group of cancers, such as sarcoma. The fact remains, however, that occasionally there is a remarkable diminution in the size of one of these sarcomas following the use of injections of erysipelas toxins. The Coley toxin is used in this way: you begin with one-quarter of a minim, as a rule, which produces no reaction. Sometimes such a small dose produces a sharp reaction in the way of a chill and an increase in temperature, with a feeling of malaise. After first trying one-quarter of a minim, you increase the dose to one-half of a minim, and then to three-quarters of a minim, and then to a minim, three minims, four minims, five or six minims. We seldom in our work use over seven or eight minims of the toxin. The idea is to produce a definite impression upon the individual as determined by an increase in temperature, a slight chill, and a feeling of malaise. This treatment is kept up for a long time in different cases. Occasionally we have standing out of a large group of cases a singularly exceptional case of great improvement or even a possible cure.

In our series of possibly seventy or eighty cases I have never seen a cure of sarcoma from Coley's fluid alone. I regard it simply as an agent which is, from our experience, capable, in a certain limited number of cases, of diminishing the size of the growth, carrying with it not a definite specific effect which we can be assured we can ac-

comply in a given case, but merely the possibility of benefit. The toxins of erysipelas, like the X-ray, should be used simply to the point where their employment is not of grave injury to the patient. Used in non-expert hands, a great many of these patients have had very great injury done to them.

In addition to these two agents there are two other phases of the treatment which I think should be discussed briefly, one of which is to make the patient physically and mentally comfortable; mentally comfortable from the standpoint of not telling this patient that his case is entirely hopeless, but telling him that his case is one in which an operation had best not be done; a case where we feel that the disease should be handled with the X-ray and with these toxins. I would certainly give him a hopeful view of his case, but I do not mean by that we should assure him that we are going to cure him, but give him this sort of hopeful view as compared with the hopeless outlook that a man would have if you were to tell him he had cancer and that nothing could be done for him. These agents give him a fighting chance. That is the statement I make in these inoperable cases. It is done for the psychic effect and for the benefit of the patient.

So far as making the patient physically comfortable is concerned, a patient like this should not be permitted to suffer any great amount of pain. If this man has pain from pressure upon nerves, from the invasion of bone, give him enough narcotics, beginning with some simple agent like aspirin when the pains are very slight; then when the aspirin does not control the nerve pain, administer codeine. When it has lost its effect, give morphine. It does not make any difference whether this man is going to take one-quarter of a grain of morphine three times a day, or a grain three times a day. In the last stages of the handling of a proposition of this kind the patient should be made comfortable, and it is your duty as a scientific physician to carry a case of this kind along to its termination. It won't do to shirk that responsibility. Somebody must do it—either you, a Christian Scientist, or Chinese doctor—and it is very much better to have these patients handled by a scientific physician so that they will be given the greatest possible benefit of comfort and relief.

In connection with cases of this kind, I want to say that we have under observation now a patient with a large sarcoma of the ilium,

which I operated upon four years ago. In that case it was not possible to remove the entire sarcoma. It was in exactly the same location, the right lower quadrant of the abdomen. The upper part of the sarcoma was cystic. We made a huge muscle-splitting incision, removed probably four-fifths of the entire circumference of a more or less globular mass that sprang from the periosteum into the iliac fossa. Why not remove the ilium? We could not take out the entire ilium, although that has been done; that is, amputation of the entire lower extremity, including the innominate bone, corresponding very much to the amputation of the entire upper portion, including the clavicle and scapula. But the results have not been at all encouraging. One-fifth of the entire periphery of this mass springing from the periosteum and ilium was curetted out with a sharp spoon. We then followed that with the use of the X-ray. The patient is still alive without a recurrence, four or five years afterward. The patient was under treatment by an X-ray man in a neighboring city who, during a period of a year or more, gave her something like one hundred exposures, as a result of which she was burned very badly. She has been back to us several times since the original operation, for plastic operations for huge X-ray burns in this locality. After a number of operations we finally succeeded in getting rid of the ill-effects of the X-ray burns, but, as I have said, there is no recurrence in this particular case. It is, however, an exception. I simply cite this case as an example of what can be done by using every means in our power to cure these apparently hopeless cases.

Before I continue this discussion, I want to show you an interesting case, which is, to a certain extent, an abdominal case, that came into our service a few days ago, and was operated on last Friday. This boy is twenty years of age and was crushed in a mining cave-in, sustaining a gross injury of his pelvis. Following that injury he was unable to urinate. Blood passed through his urethra, but no urine. A very competent general practitioner was called, a man who does a good deal of surgery, who found a distended bladder. He could not pass a catheter, and in the emergency he did a suprapubic cystotomy, and introduced a tube into the bladder for the purpose of draining off the urine. The X-ray picture showed a separation at the symphysis. There is no gross fracture of the pubic bones, but a definite marked separation had occurred at the symphysis. The boy

improved for a short time, and then began to pass large amounts of very foul-smelling pus from his urethra, but without any urine, this passing entirely from the suprapubic opening. When I examined him Friday I found he was very tender in his perineum, especially on the right side in the neighborhood of the seat of the gross injury of the pelvis. There was some induration at that point.

We are going to wash out his bladder to-day through the external urethrotomy wound that I made last Friday.

This is a very interesting problem from a surgical standpoint. I found this definite induration in the perineum on the right side. In an attempt to pass a sound we were not successful. We have here a tube which passes through the external urethrotomy wound into the bladder, and I will tell you how we did that. Here was a definite specific problem with which we had to deal. He had a suprapubic incision out of which his urine came; there was this induration in the pelvis, and a lot of pus which was foul smelling coming out from the urethra. It smelled like *fæces*. There was, however, no evidence that the rectum had been grossly injured, because he had bowel movements without any passage of urine through the rectum. Under a general anæsthetic I tried to pass a sound through the urethra, through this point of the anterior urethra, into the bladder, but just in the indurated area I came upon an obstruction and did not use any force. I simply passed a staff into the urethra so that I could cut down on that staff, and made an incision in the middle line of the perineum over the urethra. I then cut down through the skin and the superficial fascia and the median raphé of the perineum where we have the accelerator urinæ muscles coming together and keeping in front of the mid-perineal body. With one stroke after another I cut down through these structures until I reached a huge abscess. Pus began to flow out. The induration in the perineum was apparently a limited one; about a teacupful of pus escaped, and the pus still continued to flow in a much larger quantity than could have been accommodated in any pocket in the perineum itself. I then introduced my finger into the abscess and found a huge abscess not only in the perineum but also inside the pelvis, so that I could introduce my finger inside the pubic bone and still feel a great cavity out of which the pus came. The tissues were entirely unrecognizable. You could not tell the muscles of the perineum from the urethra or from anything else.

There was a big sloughing mass. What were we to do in that case? I wanted to restore that man's urethra. I wanted to obtain the drainage of his urine through the perineum. It was impossible without a guide to enter that man's bladder without doing a great deal of damage. A very interesting thing occurred to me, and I suppose it has occurred to other surgeons, because it occurred to the first professor of surgery at Rush College—Daniel Brainard. Away back in 1834 he wrote an article in which he described the operation of extroduction of a bougie. It was published in a small medical journal in Chicago, when Chicago was a town of little more than five or six thousand people. It made a great impression upon surgeons at that time, and is reported in the literature as having been the first case in which a surgeon resorted to this so-called operation of extroduction of a bougie. What is meant by that?

Brainard was confronted with a case of rupture of the urethra that had suppurated. He was a skilful anatomist and surgeon and attempted to make an external urethrotomy. He cut down on the bladder through the perineum, but could not find his way, and sometimes these external urethrotomies without a guide are exceedingly difficult. Evidently he had what we would call in a slang way a "hunch." He was stumped. He did not know what to do. I have seen men waste hours in trying to do an external urethrotomy without a guide, getting nowhere and doing much damage. In Brainard's patient the bladder was full and distended, and under an anæsthetic he did a suprapubic cystotomy. But he did not stop there. He then hunted for the internal orifice of the urethra in the bladder. He took a bougie and passed it through the opening into the bladder, then into the internal orifice of the urethra and out from that internal orifice into the perineal wound, and then passed the bougie through the external urethra, so that he could make one continuous passage of the bougie from the bladder out through the urethra. This is known as the extroduction of a bougie, and it came in very handy in this case, because otherwise, without such technic, it would have been impossible for us to have obtained perineal drainage of the bladder below without doing very great damage. Fortunately in this particular case a suprapubic cystotomy has already been done, so that all that was necessary for me to do was to enlarge the suprapubic cystotomy wound, which had contracted a little, to introduce my finger into the bladder,

feel the internal orifice of the urethra, take a sound and pass it into the internal orifice of the urethra and out into the perineum. You will understand this was very important, because the tissues of the perineum were soggy and unrecognizable. No surgeon or anatomist could have recognized the urethra from any other structure, but in this way we had a definite, reliable guide. Then I took a rubber tube, which you see in the perineum now, passed it through the bladder out through the first part of the urethra and pulled it out through the perineum, sponged out the pus and packed the cavity lightly with iodoform gauze. I then sewed the tube in the perineum with a silkworm-gut suture so that it would not slip out. I was careful to leave that much (indicating about one inch) of the tube inside the bladder. If I had left six inches of the tube inside the bladder it would have curled up and would not have drained. As it is, the tube projects well into the bladder, and at the same time it is not long enough to curl upon itself and interfere with drainage.

What is going to happen in this case and what is your definite surgical problem? You have here to deal with a rupture of the urethra plus a gross infection which has made a huge abscess both in the perineum and the pelvis, associated with more or less sloughing of tissue. A bacteriologic examination was made, and, as you might imagine, there were staphylococcus and colon organisms. I told you the pus smelled like feces. There is no evidence that there is a lesion of the rectum communicating with this space, but the conditions here are such as to make it easily possible for colon infection to occur because of the proximity of the rectum to this large traumatic wound. The fact is that colon infections are among the most common infections associated with infections of the bladder and of the urethra. Our immediate problem is to drain the abscess and to drain the bladder. On Friday we operated. At that time the patient had a temperature of 103° F.; the next day the highest temperature reached was 101°, and it has not been 100° since. The fever is coming down in a very satisfactory way. The patient had a great deal of pain before the operation because the pus was under great tension. That pain has disappeared and his appetite has improved. The immediate indication was the establishment of good drainage of the abscess and the establishment of thorough drainage of the urine through the perineal incision. But that is not enough. This boy will

undoubtedly recover from the immediate effects of this injury and of the infection, but unless we carry him along in a scientific way, in a proper surgical way, he will be very much damaged. He would undoubtedly have, as a result of this large abscess, a traumatic stricture of the urethra, and a stricture which, because of the associated traumatism and the infection, would contract rapidly and lead to great urinary difficulty. How are you going to handle this case? Can you do a plastic of the urethra here? Certainly not. We very often discuss learnedly plastics of the urethra, finding the ends of the urethra and sewing them together, and repairing the urethra in that way. You can not recognize the urethra in this case unless you determine the position with extrodution of a bougie and determine the position of the distal end of the urethra at the site of rupture by passing a bougie through the urethra from in front. No plastic operation would be of any service here. It could not be done anatomically, and could not succeed in the presence of this suppuration and sloughing. You can not consider that plan at all. What can you do then in the handling of this case, not only to get the boy well from the immediate effects of the injury, but to keep him well so that he will have a urethra of fair calibre, which will functionate and carry him through the rest of his life without giving him any great amount of difficulty? I dare say that, as a result of the injury in this case, we might lose an inch of this man's urethra from the traumatism, plus the infection and the sloughing. What is going to take place? How are you going to restore an inch of that urethra? Let us take that as a concrete problem.

Within a few days or a week this large abscess cavity will gradually close down. The space will be filled by granulation tissue. You will have a tube in the proximal part of the urethra, extending from the bladder out through the perineum. Your problem will be this: to maintain in the wound repair a urethra of large calibre at the point where the urethra is gone, or rather a canal of large calibre at that point where the urethra is gone, and where you have, say, an inch of granulation tissue. That will repair itself if you maintain a sufficiently large canal between the two ends of the urethra. You have to maintain the canal between the proximal end of the urethra and distal end where that space is covered in by the granulation tissue. Gradually, in the process of the repair and in the management of this

case, we shall at the end of a few days remove the perineal tube, and there will be a tendency for the perineal incision gradually to close. We shall then pass large sounds from the external urinary meatus through the urethra into the bladder. This will be done possibly every other day, or every third day, so as to maintain the canal. The perineal incision will heal up entirely. From the ends of this urethra the squamous-cell epithelium will grow from this end and that end (indicating) and eventually line the connective-tissue tract formed by granulation tissue. It is very important for us to maintain a canal of large calibre, probably twice or three times the size of the normal urethra, and that is not difficult to do, because if you pass a No. 30 sound in this position it will stretch open that granulation tissue very much larger than the actual size of a No. 30 sound. These sounds will be passed every third day; gradually the perineal wound will heal up, and then he will begin to urinate through the urethra, and at the end of a month he will have a urethra of good-sized calibre. I believe I would do a little more than that: I would use sounds from 30 to 34 French. If we do that, we will succeed in securing for him a pretty good functioning urethra. It will not be a normal urethra; it will be one with a good big scar in it, and that scar always has a tendency to contract.

This patient for a long time should be kept under observation. While he is with us we will pass these sounds frequently, and then send him home and instruct his medical attendant in his town for a period of three months to pass a sound every week. In order to make it comfortable for the patient, this ought to be done with novocaine, so that the urethra will be anæsthetized locally. It ought to be done gently as well as expertly. If at the end of three months there is a little tendency to contract, the wise attending physician will tell this boy to come to him once a month. If this continues for the next year, he will tell him to report once in three months. You see what a long-drawn-out course of treatment a case such as this requires. He must be told that, if there is any tendency at all during the rest of his life to interference with urination, to report at once. I would like to have all of you have that mental conception of this wound healing in this particular case, what really happens, and how you could influence the wound healing in order to secure the best possible functional results.

This morning I want to spend the last twenty minutes of our time in outlining some of the general principles in abdominal surgery. Referring again to the clinical problem that will confront you whenever you undertake the management of an abdominal case, I want to say it is necessary for you to study exhaustively a case, first, from the standpoint of diagnosis, and I am going to use the word "science" of diagnosis, and, second, from the standpoint of the *science of surgical therapy*.

From the standpoint of diagnosis, there are certain pieces of evidence which you must detect, and I want to divide these pieces of evidence under four heads: *First*, The history of the case, and the evidence of course obtained from that history; *second*, evidence obtained from physical examination; *third*, evidence obtained from a laboratory examination; *fourth*, in abdominal surgery we make a special division of the evidence obtained by the X-ray.

Before proceeding any further, I want to give you a general impression of the value of these four pieces of evidence in a particular case, or rather as applied to a large group of cases. I want to give you an estimate of the value of this evidence, because I think it will be of service to you in your actual work. Attempting, as we have done in our service, to look upon these cases as pieces of clinical research, and analyzing them from that standpoint, we have estimated the value of these four different pieces of evidence, and the conclusions that we have arrived at are that the history, if properly taken, covering the whole ground, and properly interpreted, furnishes you, as a rule, much more than one-half of the evidence upon which you base your diagnosis. I want to emphasize very forcibly that much more than a majority, so far as the value of the evidence is concerned, in making a diagnosis in an abdominal case, or, for that matter, in a surgical case, much more than a majority of the evidence, so far as its value is concerned, is obtained from a carefully prepared obtained history, properly interpreted. Estimated in figures, 25 per cent., or half the balance of the evidence, is obtained from physical examination. The other 25 per cent. is pretty well divided in abdominal work between the evidence obtained in the laboratory and the evidence gathered in the X-ray room. Figures of this kind can not be limited to a specific single case, because in certain cases one piece of evidence is absolutely determining. A man comes here

with a history of pain in his lumbar region, of having had so much pain that he has to have a hypodermic injection of morphine to control it. It may last for a few hours; he passes a lot of blood through the urethra, and an X-ray picture shows a stone in his kidney. The X-ray would be absolutely determining, and one should not say the history represented more than one-half the value of the evidence in such a case; but I am taking a group of abdominal cases, say a thousand abdominal cases.

The same thing is true of some of our laboratory findings. A patient comes in here for me to do an abdominal operation for a large abdominal tumor. In examining the patient I find a large tumor on the left side as big as a child's head. I get a drop of blood and put it under the microscope and find the typical, multiform picture of splenic leukæmia. The laboratory test in such a case is very determining. But I have expressed these figures to you as applying to a general group of cases. Therefore, whenever we undertake the handling of an abdominal case, the first thing to do is to get a very exhaustive, clean-cut history of the case. A good and satisfactory history of a case is largely a matter of personal equation. That personal equation is determined very largely by the training that the man has had who has taken the history and the experience he has had. I am not in favor of elaborate histories, with a dozen or forty or one hundred different topics printed out in the obtaining of a history. I like to go at it in a simple way. I like to take a good, careful history of the patient, not an exhaustive one, and also the family history. Very often you will find a rather finicky medical man spending altogether too much time on unimportant details in regard to the family history. I do not care very much for an exhaustive family history unless, in the after-development of the case, it seems desirable to obtain it. It is well to go very rapidly to the patient's own condition, his previous illnesses and injuries, if any, and then, without too many leading questions. Still, it is necessary to keep the patient in a groove, more or less narrow. You come at once to the specific thing for which he comes to you, or for which she comes to you, as the case may be, and with this history briefly stated I go back and ask for evidence very definitely in the previous history which points to the condition of which the patient complains. Of course, in some cases we are confronted with facts which we can not control. An

accurate history is very difficult to obtain. In the case of an adult sometimes we find in our hospital work, particularly in cases of foreigners, it is difficult to obtain a good, reliable history. This is particularly the case in individuals who are comatose or who are irrational. Very often it is difficult for us to obtain an accurate history in cases of this kind; nevertheless, I want to emphasize the importance of an accurate history.

Secondly, the question of physical examination. I want to dwell a little on this point. A physical examination, to be of any value, must be made by some one who is competent to do it. A physical examination in the hands of an expert may mean a great deal, while in the hands of a tyro it may mean little or nothing. Let me give you just one concrete example. When one first begins to make a physical examination and a bimanual examination of the female pelvis, the first impressions are very vague. It takes a certain amount of training, repeated examinations, and instruction as to the interpretation of the things that are found to make that examination of much value, and to make it of the greatest value one must imagine that the individual has made dozens, and even hundreds, of these examinations, not only in normal individuals but in regard to a variety of pelvic conditions, and has learned how to interpret the findings which he obtains. When one first makes a physical examination of the female pelvis it is difficult to outline the uterus. The cervix is felt, but it is hard to outline the uterus accurately. With some experience the uterus can be outlined; with still more experience the right ovary can be felt, and with a little more training the tube can, as a rule, be outlined. The same thing is true of the left ovary. With still more experience the individual is able to say, when he makes a bimanual examination, here is a mass attached to the uterus or to the ovary, or here is a mass that is not connected with the uterus or with the ovary at all. It is a mass that occupies a position in the sacral fossa, or it is a mass in connection with the cæcum, or a mass in connection with the lymphatic glands in the pelvis. Now, what I want to emphasize in connection with a physical examination is that this examination is exceedingly important; that it means a lot of training; that that training you must get here in your dispensary work in the college; that it is carried on in your work as an intern, and that even then you will simply begin to have a conception of

how to make a physical examination. Then later, as you gain wider experience, the whole field opens up to you, so that as an expert you can put an abdominal case on the table, make a bimanual examination, and feel accurately everything that is in the pelvis. You will be able to feel the cæcum, the right kidney, the sigmoid, and very frequently the pancreas. As your fingers touch the field under examination you have a sense of assurance which increases with practice. It is a matter of training and experience, but what I want particularly to emphasize to you is that that sort of training and experience is scientific. It is so important that it overshadows laboratory work, so far as its numerical value is concerned as applied to scientific surgery; that it is not to be belittled, and that it is one of the most important things you can do in developing yourselves as expert clinicians.

Just a word in regard to laboratory work. Modern scientific surgery has taken into its territory everything that can be utilized in the way of laboratory work. Laboratory work is absolutely indispensable in doing good abdominal surgery. A routine laboratory examination of the urine, of the fæces, of the blood, the many laboratory tests, such as the Wassermann and the various bacteriologic tests that we make in the laboratory, are absolutely essential to the doing of good scientific work in abdominal surgery. One must, however, in analyzing this subject, realize the element of error that creeps into all the different pieces of evidence that we are seeking to obtain. I want to pause a moment to consider this, because I find most of our students and most of our young physicians, with very good laboratory training, are inclined to regard many pieces of laboratory evidence as infallible and to allow those pieces of evidence to overshadow other conditions in the cases. I find this repeatedly in our surgical work, and I would like to have you come to a more judicial opinion and to place yourselves in a more judicial attitude toward these various laboratory tests. For instance, we have an abdominal condition to deal with; we make an examination of the urine and find some blood-corpuscles in the urine. The patient has considerable elevation of temperature and acute abdominal pains. Frequently we have found that if we had relied as a determining factor on the presence of blood in the urine, and had come to the conclusion that we had to deal with a lesion of the urinary tract because of that finding, we would have made a

gross mistake, because we find not infrequently in an acute appendicitis red blood-cells in the urine. Let me cite a case.

A man, forty years of age, with a very severe acute abdominal attack, referred especially to the lumbar region, was watched by a very able surgeon and good internist for forty-eight hours. Examination of the urine showed the presence of a definite number of red blood-corpuscles. The attack was short. Within 24 to 36 hours the pain had disappeared, the temperature had fallen, and the case was diagnosed as a kidney lesion with hæmaturia. Then there was a chill, another elevation of temperature, and a few days later I opened a huge appendiceal abscess well back in the flank. The after-history demonstrated that there had been no lesion of the kidney at any time. In other words, if we had allowed ourselves to be governed by the presence of blood in the urine in that particular case and accepted that as the determining factor, it would have been a gross clinical error. A great many hundred cases of acute appendicitis show that it is not at all an infrequent finding.

The same is true of the Wassermann test. I will leave the field of abdominal surgery for a moment and apply such a laboratory test as the Wassermann to a lesion of the tongue as we find it is very frequently applied in actual clinical work. Time and again a patient comes to us with a lesion of the tongue, with a history that a Wassermann test has been made and found to be strongly positive; that it was a syphilitic lesion, and the patient had been kept for three or six weeks or six months on antisyphilitic treatment, and unfortunately was no better. What a gross error it would be if we looked at the laboratory test alone and relied on it! What are the facts? The facts are that syphilitic lesions of the tongue are the most frequent etiologic factors in carcinoma of that organ. Of course, you may have the report of a positive Wassermann, and if you relied on the Wassermann test alone you would be frequently in error, and the patient, because of your lack of knowledge, would lose the opportunity of having a radical operation done at a time when there was some hope of a permanent cure. One can not place too high an estimate on the value of laboratory findings as applied to abdominal surgery; but I want you to remember, in your estimate of such findings, that they are to be taken into consideration with the other evidence at hand, and with a very broad estimate of the other evidence at hand, and

knowledge of it before a final conclusion as to the diagnosis is made.

Finally, I will spend a few moments on the last bit of evidence we employ in abdominal surgery, and that is the X-ray. Almost as soon as Röntgen announced his discovery, the X-ray was appropriated by medical men and utilized, and has been developed and is developing into one of the most important means of diagnosis that we have. At the same time, an enormous amount of injury has been done by men who have relied too much upon X-ray evidence, and who have not been in a position to estimate its value in the sum-total of the evidence obtained in a particular case. A very great amount of unnecessary surgery has been done because of the misinterpretation of the X-ray findings. Hardly a week goes by that some patient is not brought into my office with an X-ray picture of the colon or of the stomach being out of position, with the statement that Dr. So-and-So, a general practitioner, or an X-ray expert, finds that this colon is a low-lying colon; that it should be tucked up into position; that the stomach is too low and should be hammocked up; that the cæcum is too big and too voluminous and it should be reefed; that the ileocaecal valve is not competent, and that some operation should be done on it—all of these things based on X-ray evidence. As a matter of fact, it is evidence that has been poorly studied from a scientific standpoint and not properly interpreted.

When we began to use the X-ray as a means of diagnosis in abdominal surgery we were greatly surprised to find that the normal position of the stomach was two or three inches lower than we used to think it was; that it was away down sometimes in the false pelvis. When we began these studies we had a sort of fixed idea of what was the normal relationship of the large bowel to the rest of the abdominal contents, and we drew pictures of a cæcum passing up into the ascending colon and shooting straight across the abdominal cavity and holding up the splenic flexure and going straight down on the left side into the descending colon, sigmoid flexure, and rectum. After we had studied a large number of these X-ray findings we found that our preconceived ideas of the anatomy of the colon were not correct, but that frequently the individual was perfectly normal, and had a transverse colon that went down into his or her pelvis; that very frequently we had a perfectly huge cæcum without an abnormality, so far as the life of the individual was concerned. We found such a piece of

evidence as this: in a neighboring city or state a very good X-ray man radiographed a number of patients, injected the colon with barium, and in a certain number of cases without much pressure he found there was regurgitation from the ileocaecal valve into the first six or eight inches of the ileum. What do you suppose he did? Instead of studying the problem in a scientific way, he at once jumped to the conclusion that these particular cases had insufficiency of the ileocaecal valve. Most of these patients came to him with the idea that there was some abdominal distress. The ileocaecal valve was patulous, therefore insufficiency of the ileocaecal valve is the cause of the abdominal distress, and in one year he did 128 laparotomies for insufficiency of the ileocaecal valve.

A careful analysis shows that if we would take this group of individuals who do complain of any abdominal distress, or take individuals who do not complain of any abdominal distress, and fill their colons with the barium solution, there would be a considerable number in whom the barium would go back into the ileum without any clinical evidence or history of any trouble whatsoever.

For a time the X-ray was given altogether too much credit as an infallible aid in making a diagnosis in abdominal lesions. Fortunately in the last few years a very great deal of careful study has been devoted to the subject, and there is no single piece of evidence that is of greater importance in a large group of abdominal cases than the X-ray, properly managed and where the evidence is properly interpreted.

**SURGICAL CLINIC OF DR. FRANK H. LAHEY BEFORE
THE FOURTH-YEAR STUDENTS OF TUFTS
MEDICAL SCHOOL**

AT BOSTON CITY HOSPITAL

Adenoma of thyroid gland (intrathoracic)	Chronic mastitis
Exophthalmic goitre	Inguinal hernia

Adenoma of Thyroid Gland (Intrathoracic).—The first case this morning is a woman, married, forty-two years of age. For the past eight years the patient has noticed a small swelling in the front part of the neck, low down on the left side, which has grown harder and larger; it causes no pain and is not tender. Of late, upon eating, food has not seemed to go down, and when she coughs her “wind is shut off.” She has palpitation of the heart on slight exertion, as climbing stairs, and is very nervous. Her hair has been falling out. Her mouth and throat have been dry of late. She does not perspire easily; her skin has been hot. Appetite is good; no distress after eating. No jaundice, no constipation, no cough, no hæmoptysis, no night-sweats. Four pounds loss in weight. She has never had œdema of the extremities except when pregnant. Her sister has noticed a change in her voice. Nocturia, once; polyuria during the day; no dysuria; no hæmaturia. Menses started at fifteen; always regular and not painful; lately the flow has been scanty. She has given birth to eight children, all living and well, the youngest seven years old; no instrumental deliveries, no miscarriages. Her family history is unimportant as concerns this trouble. She has had measles, typhoid fever, and scarlet fever, otherwise has been well; never had tonsillitis, denies venereal disease in any form. She works all day and sleeps well. Her general physical examination is negative except for the local examination of her neck. Her pupils are equal and regular and react to light and distance. There is a slight exophthalmos. Both eyes converge. No diminution in palpebral fissure. No von Graefe’s sign. No increase in frequency of winking. Fine tremor of hands; no tremor of toes.

We think that this woman has an adenoma of the thyroid, origi-

nating at the lower pole of the left lobe, and that about one-half of the adenoma is intrathoracic in location. It is probably about the size of a tangerine orange. She is being operated on for the following reasons:

1. Because she has slight pressure symptoms;
2. Because she has slight symptoms of secondary hyperthyroidism;
3. Because her adenoma is partly intrathoracic in location; and
4. Because of the danger of malignant degeneration in adenomata of the thyroid.

She has hyperthyroidism to a slight degree, as evidenced by the slightly prominent eyes, by the fine tremor, by the tachycardia on exertion, by the loss of weight, and by the nervousness. This is secondary, not primary, hyperthyroidism.

Of the two types of hyperthyroidism, primary and secondary, the former, or true exophthalmic goitre, is the type which originates in a symmetrically enlarged thyroid, irrespective of any new-growth. It comes on with no apparent cause. The thyroid is symmetrically enlarged, the right lobe usually being a little larger than the left. It is more intense, as a rule, than secondary hyperthyroidism; otherwise, so far as symptoms go, they are just the same. Secondary hyperthyroidism is a condition of hyperthyroidism which occurs because of, and following, the appearance of an adenoma of the thyroid gland. The symptoms are just the same as those of primary hyperthyroidism, the condition differing only in cause. It is probable that the cause of hyperthyroidism in primary hyperthyroidism is an activity of nearly all the thyroid gland, while the cause of secondary hyperthyroidism is probably an activity of the thyroid cells within the adenoma. The treatment, therefore, of the two conditions is entirely different. Primary hyperthyroidism, in which the whole gland is activated, must be treated by the removal of a large portion of the thyroid gland, so that the remaining portion will secrete or functionate to the amount corresponding to that of a normally functioning thyroid. Hence in primary hyperthyroidism one lobe, the isthmus, and usually a considerable portion of the remaining lobe, must be removed. On the other hand, in secondary hyperthyroidism it suffices to remove the adenoma, and with its removal it is probably true that the overactivating cells are removed, those being the ones

that are in the adenoma. It is a fact, at any rate, that secondary hyperthyroidism is benefited or cured by the simple removal of the adenoma, the gland itself being untouched.

We are now going to follow our general rule and operate upon this woman with local anæsthesia, combined with preliminary drugging. Local anæsthesia is not sufficient to do away with all of the pain. The anæsthesia is not sufficiently complete, so that some of the painful stimuli do not penetrate. If they do penetrate, the patient is excited, apprehensive, anxious, and the whole condition is thoroughly undesirable in the treatment of thyroid diseases. In these cases, therefore, there must be an addition to local anæsthesia; namely, preliminary drugging. Our method of preliminary drugging is scopolamine and morphine. We give these patients, two hours before the operation, one two-hundredth of a grain of scopolamine and one-fourth of a grain of morphine hypodermically. At the end of another hour, provided no untoward symptoms have occurred—and in a series of over 140 cases there have been no untoward symptoms—we repeat this dose, giving another one two-hundredth of scopolamine and one-quarter of morphine. At the time of operation the patient is observed, and, if soundly sleeping, no more drugging is used; if, however, still awake, one four-hundredth of a grain of scopolamine and no morphine is given. This means that approximately one-eightieth of a grain of scopolamine is given in two hours. Unfortunately, scopolamine is irregular in its action; some patients are susceptible to it, others not. I should say that seven or eight out of ten patients react properly—what we consider properly—to scopolamine; that is, go to sleep and are not apprehensive throughout the operation, and in no way interfere with the procedure under local anæsthesia. Unfortunately the three others do not; of these some are slightly drowsy, others wide awake. In such cases it becomes necessary to use some other form of anæsthesia in addition. It has been our custom at such times to administer nitrous oxide, particularly if the patient is apprehensive or anxious, or while we are pulling on the thyroid gland or dislocating an adenoma from the chest. We therefore always have ready a gas-oxygen anæsthetist in case the patient wakes up or becomes anxious or is suffering at all from the painful manipulation from dragging upon the gland.

As regards the local anæsthesia, the percentage of novocaine solu-

tion is two per cent. with 15 minims of adrenalin to the ounce. We can use up to two ounces of this solution, and in some cases more. It is injected into the skin, into the platysma and subcutaneous fat, and along the borders of the sternomastoid and in the median line where the sternohyoid and sternothyroid join. Beyond this, novocaine is unnecessary, as manipulation of the thyroid gland is not painful except when it is dragged upon; cutting the thyroid gland does not produce pain, nor does the introduction of hooks. If, however, it is pulled upon, considerable pain is produced. One other point in regard to scopolamine and gas: But a very small amount of gas is needed to produce deep anæsthesia in these scopolaminized patients—just a whiff or two of gas will often set them to snoring; another interesting fact is that, although they be wide awake and apprehensive, once you get them to sleep with nitrous oxide you can take off the nitrous oxide and they will remain asleep a long time, but if you wake them by a painful stimulation it may again become necessary to give the gas.

(The patient is brought in and infiltration begun.)

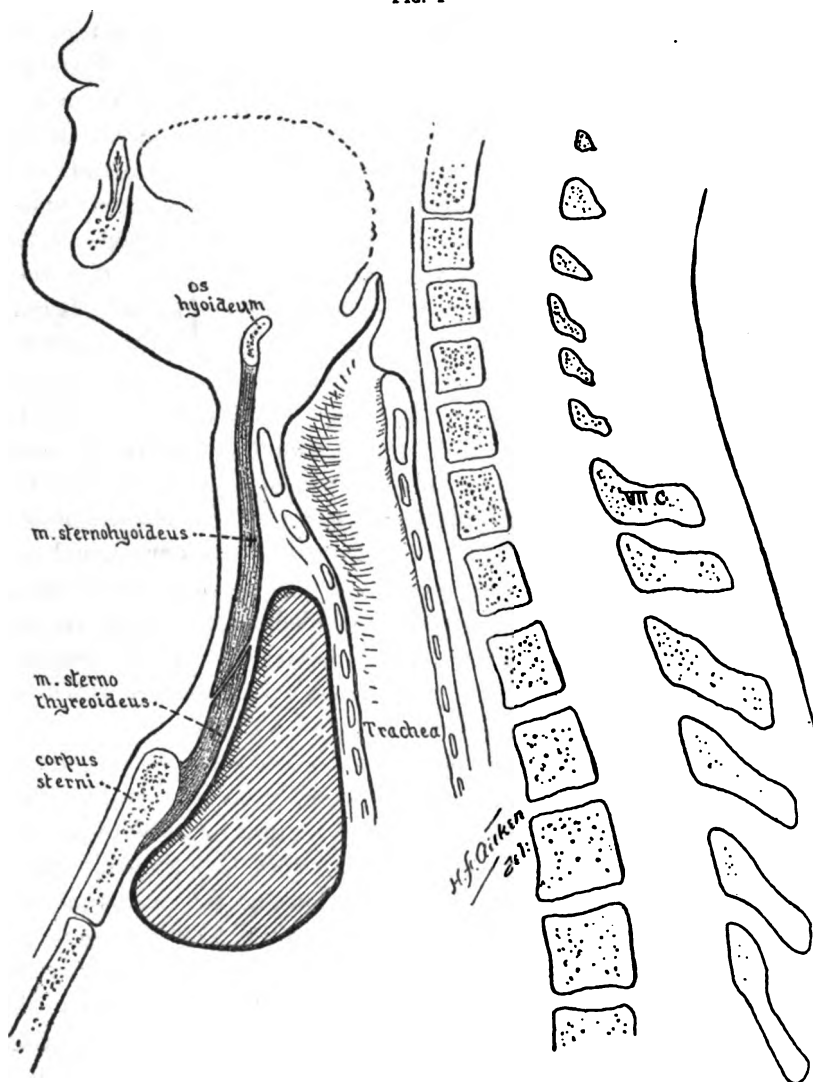
You will notice the white wheal which appears at once with the proper introduction of the novocaine solution into and not beneath the skin. This is the so-called collar incision. Notice that it is not easy to get the proper introduction of the novocaine between the skin. The skin is thin and the needle must go directly into it. With the proper injection of the novocaine solution, we can feel the distention of the skin between the thumb and index-finger. The next infiltration is into the platysma and subcutaneous fat. (Incision made.) This is the advantage of two per cent. novocaine solution: you do not have to wait for anæsthesia; you can begin your incision at once. The platysma and skin, you can see, are separated by blunt dissection at first and along the line of the marked-out incision. We will now tie these small veins beneath the skin so as to get them out of the way and turn the flap up. We now dissect the skin and platysma. Here are the large anterior veins of the neck, which we try to avoid. There is the edge of the sternomastoid. This is the edge of the sternomastoid on the other side. This is a small incision, because the adenoma lies near the middle line, and we think we can get at it easily. You will notice there is very little goitre to be seen, yet you will be surprised at the size of the adenoma when it is taken out of the thorax.

(Success in these thyroid cases under local anaesthesia depends on gentleness of manipulation; there is no place for hasty pulling and hauling in this kind of surgery.) We now introduce novocaine along the anterior edge of the sternomastoid on either side, and in the middle line where the sternohyoid and sternothyroid join. We separate the sternohyoid and sternothyroid on either side at the middle line, and then come directly to the goitre. We separate the sternohyoid and sternothyroid from the anterior surface of the gland itself by blunt dissection with the finger until the finger is well out under the edge of the sternomastoid. Next we separate the edge of the sternomastoid from the sternohyoid and sternothyroid so that they may be clamped and cut well out beneath the sternomastoid. It may be possible that I can get along with cutting the muscles transversely on but one side, although there is no point, I believe, in being handicapped in these goitre operations by lack of exposure. Here is the thyroid and below is the capsule of the tumor. This is the notch of the sternum, and the remaining portion of the tumor is in the thorax. The tumor is sufficiently large so that I will cut the muscle on the other side. On this side I again separate the sternohyoid and sternothyroid and cut between clamps as on the other side; notice that the large anterior veins will be included in the clamp, so that bleeding will be taken care of. The goitre is now well exposed except for a few muscle-fibres over the anterior surface which we will separate, and here is the adenoma.

My finger here goes well down into the thorax; in the delivery of intrathoracic adenomata it is first necessary to be sure that they are entirely freed well down to their lowest limit; otherwise, if they are dragged out of the chest, small bleeders may be left inside which produce hæmatomata and result in infection and serious pulmonary conditions. This runs fairly well down into the chest. One must get into the right layer of these adenomata or they will not deliver. It is going to be difficult to deliver, because it is firmly adherent to the trachea. This is what hurts (the pulling). These adenomata all have a capsule which consists of dense connective tissue; it is due to the effect of the adenoma acting as a foreign body in the gland. In fact, the adenoma is a foreign body; it becomes surrounded with connective tissue just as any foreign body, and with its expansion this connective tissue becomes condensed until it produces a definite

capsule, which you can see plainly now under my scissors. There is the capsule, and now I think I shall be able to deliver this tumor;

FIG. 1



Showing how adenomata of the thyroid become intrathoracic in location. Note that as the lower pole increases in diameter the upper pole will be dragged downward until it may eventually disappear into chest.

no, it is so firmly adherent that I shall first have to dissect it from the trachea. Now it begins to come up a little bit better, but the adhesions are very firm. You notice the effect of pulling, producing

a cough. These adhesions are so firm that delivery is quite difficult. At last we have the lower pole delivered and can begin to clamp its blood supply off. You see, this is the first real pain she has suffered, and this is due entirely to dragging on the tumor. This will be over in just a minute. The tumor is now being excised from true thyroid tissue in which it rests.

There is the adenoma, now completely freed. I think you can see just how the adenoma lay in the chest. See Diagram I, illustrating how adenomata become intrathoracic, and Fig. 2, showing tumor after removal (note pear-shaped intrathoracic portion).

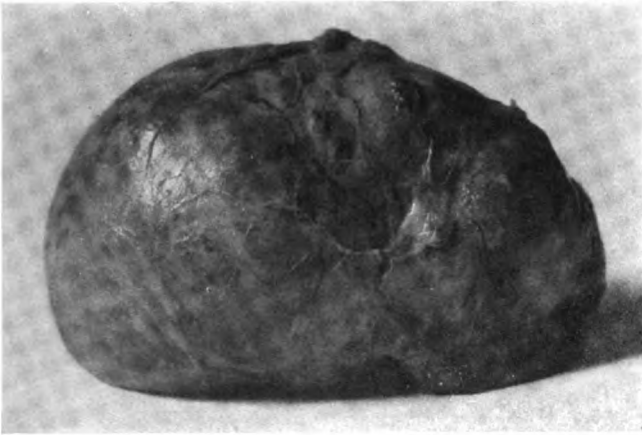
This diagram shows how intrathoracic adenomata must become pear-shaped eventually. They will become pear-shaped with the larger end down for the following reasons: once the adenoma gets into the chest, the funnel shape of the chest always permits the lower portion to expand, while the upper portion is contracted by the narrow thoracic aperture, so that the portion which is above may be only a small nodule, while that portion in the chest may be of considerable diameter. As the intrathoracic portion enlarges, it must taper toward the top by drawing down the extrathoracic part. Often the adenomata, which are located largely within the chest, are considered of no importance by the family physician or by the patient because the part that is visible is but a very small portion, and the part that is intrathoracic is overlooked.

These vessels must now be ligated securely to avoid the danger of secondary hemorrhage. Notice the depth of the hole from which this adenoma is delivered. The bleeding is now all controlled, and we will restore the neck by suturing the cut muscles. These are sutured with mattress sutures to grasp any of the veins on or in the muscles and control any possible bleeding from the cut ends. (You will notice that these needle-holders are copper-plated, which is for the purpose of distinguishing them from the other instruments which closely resemble them and are nickel-plated.) These muscles are now sutured together and the flap is restored; first, the platysma is sutured with interrupted stitches to prevent spreading of the wound, and then the skin with horsehair and an intestinal needle—that is, a round needle. Too much care can not be exercised in suturing the wound for accurate approximation, inasmuch as the scar is practically always visible in women.

In order that you may obtain a correct impression of how much patients suffer with this method of preliminary drugging and local anæsthesia, I shall ask the patient when I get through to tell you honestly just how much she suffered. The patient says, "Nothing," and that she felt it only once when I was pulling it out.

Exophthalmic Goitre.—The next case is a primary hyperthyroidism or exophthalmic goitre. She is forty years of age, widow, born in Nova Scotia. For the last few years the patient has felt run down and weak. That is a frequent history with exophthalmic goitre cases; they are run down and weak, or suffer from nervous collapse or some similar condition, which is later found to be due to hyperthyroidism. She says she seems to have no strength at all. She has done her own housework and worked hard to take care of her husband, a paralytic. She cries easily and "trembles all over." She has palpitation, and on the slightest exertion becomes dyspnoic. At times her flesh seems to be burning all over. Her skin itches and she perspires very easily. All these are typical symptoms: the itching skin, the subjective feeling of heat, the frequent and free perspiration. She has night-sweats even in cold weather. During the last few years her hair has been falling out, and recently it has turned gray. Her friends have noticed a bulging of the eyes, though she was not aware of it herself. Strange to say, many of these cases do not notice exophthalmos until their attention is called to it. Her people say her voice has changed. Recently she has noticed a small swelling in the neck. Her appetite has been always good, and of late has increased. The metabolism is increased in hyperthyroidism, and the appetite must likewise increase to keep up with it; unfortunately the activity of the metabolism is so great in the toxic stages of exophthalmic goitre that patients can not eat enough to keep up with the overactive metabolism, with the result that they lose weight. But in practically all toxic cases of exophthalmic goitre you will find that the organism makes an attempt to keep up with the additional burning up of material by increased appetite. She eats large meals and eats very much between meals, yet, notwithstanding this fact, she has lost twenty-two pounds in the last eight months. She has difficulty in swallowing food, and states that she has to take a drink of water to wash the food down. The bowels had moved regularly until recently, when

FIG. 2



↑ Intrathoracic portion ↑
Showing the adenoma after removal. Note: The bulging lower end between the two arrows represents the intrathoracic portion.

FIG. 3



Microphotograph shows a duct greatly dilated and almost completely filled with alveolar and solid masses of tumor-cells. Notice in the secretion numbers of endothelial leucocytes, most of which contain fat or pigment. While the duct is filled with the new growth, there is no tendency to go beyond the basement membrane.

she suffered with diarrhoea, from ten to twelve movements a day. This, too, is characteristic. She has polyuria, no dysuria nor hæmaturia.

Menstrual history: Since eighteen, and always regular until the last few years, when intervals have occurred of two to three months. That is another characteristic of hyperthyroidism. She has never been pregnant, as she does not care for children; no miscarriages. During the last few months her breasts have become painful and large, a not uncommon occurrence in hyperthyroidism. No history of goitre appears in her family, and her past history is unimportant. Her pupils are equal and react to light and accommodation. There is a slight lagging of the lids, a definite widening of the palpebral fissure, and a well-marked lack of convergence in the right eye. She has a small diffuse swelling, soft and symmetrical, evidently an enlarged thyroid. She has distinct œdema of the left ankle. There is a fine tremor of the fingers and toes. This woman has a true primary exophthalmic goitre. She has had scopolamine and morphine in the same amounts as the other patient.

We shall remove one lobe, the isthmus, and part of the other lobe. In these toxic cases more than half of the thyroid must be removed—one lobe, the isthmus, and from one-fourth to one-half of the remaining lobe. In these cases a well-marked benefit and practically a complete cure may be expected.

In the facial expression, the anxious, apprehensive, drawn, excited countenance within a few days will be replaced by a calm, peaceful expression. Within two or three months a well-marked gain in weight will appear. Within two weeks there will be a well-marked diminution in pulse-rate and nervousness, and excitability will be markedly diminished within a month, and, in the successful cases, entirely lost. There is a complete change in the personality of the individual; in fact, I have repeatedly said in regard to these cases that there is no operation in surgery which gives me such satisfactory and immediate results. There are no patients whom I operate upon whom I would rather see returning than these cases of exophthalmic goitre which have been subjected to partial thyroidectomy. I am always glad to see them, because they look and feel so much better.

As you will see, it is a different operation from the removal of an adenoma. These glands are vascular, and the hemorrhage is apt

to be profuse unless the vessels are carefully ligated. The novocaine is introduced as in the previous case. Inasmuch as we must expose the superior thyroid vessels, the incision must be longer and the ends of the incision turned up so that a larger flap may be obtained. The patient is now soundly sleeping and snoring. This illustrates very well to you the variability in the effect of scopolamine: this woman had the same dose as the previous case and is snoring; the other was wide awake during the entire operation, but was not hurt except when the tumor was pulled out of the chest. We turn the external portion of the incision upward in order that we may easily get at the superior thyroid vessels. The next injection is made into the platysma and subcutaneous fat. For me this is the ideal anaesthesia for exophthalmic goitre. The woman is asleep, is unapprehensive, and her pulse will go down while the operation is being done. She is now asleep and will sleep for about six hours after the operation, and will be practically free from the psychic shock that plays a great part in the mortality of exophthalmic goitre.

You will observe the difference in vascularity even in the skin incision. Notice that the veins of the neck are not adherent to the platysma, but to the sternohyoid and thyroid. We will try to dissect this flap off the anterior veins without cutting them, though sometimes it is impossible. Now the anterior muscles of the neck are well exposed. We will infiltrate the edge of the sternomastoid with novocaine. The anterior edges of the sternomastoid are now infiltrated, and also the middle line. We dissect off the edges of the sternomastoid so that we may cut the longitudinal muscles well under them; then separate the muscles in the middle line down to the thyroid. This dissection is carried down to the surface of the gland. There is the thyroid—you can see the veins on its anterior surface. The muscles are separated from the surface anteriorly, then on the opposite side. I can now show you the thyroid gland plainly; there is the lobe on the left side, and there is the gland with a few muscle-fibres on this side over the anterior surface.

We will now remove this lobe, the isthmus, and half of the other lobe. That is the superior thyroid artery and vein; we must be careful that none of them gets left behind. It is very much better to ligate the superior pole of the thyroid first, to start the thyroidectomy at the superior pole rather than the inferior, because the gland peels out of

the capsule very much better in this direction, as I shall show you. You can now see the gland coming out of the capsule. The parathyroids and recurrent laryngeal are left behind. We put snaps on the edge of the capsule so that it is always demonstrable. Here is the small vessel running between the capsule in the gland, which is clamped and cut. We next begin to roll the gland inward so that the posterior surface is exposed. There are the branches of the inferior thyroid artery, the capsule having been wiped off and left behind intact. (Patient groans.) Again it is the dragging which causes the pain. There is the inferior thyroid vein, and we are now ready to remove the gland, having controlled the main vessels; a few on the trachea will have to be clamped, as the isthmus is dissected from it. Here is the trachea, and the gland is now dissected over to the other side; it is now clamped at the isthmus by a half-curved clamp which controls the bleeding. The isthmus is now sutured over and over with plain catgut. The vessels are now tied; they are very delicate and will not stand any pulling whatever. That is the inferior thyroid artery tied in two places, both its branches, never as a trunk. By tying the branches we avoid the recurrent laryngeal nerve. (Patient groans and moves and is given some nitrous oxide gas.)

Observe how quickly this patient goes under the influence of the gas. There is one lobe and the isthmus out. We will now remove a portion of the other lobe. The superior pole is pulled up with double hooks. The superior vessels are exposed and tied. Here is the upper pole of the gland well exposed and wiped out, and all of the blood supply controlled. We next clamp off what we think is enough of this gland and cut it away. This portion of the thyroid will drop back into this space. Observe how a single administration of gas has carried her on through this; that is, we have succeeded by the gas in getting her back to sleep again. Note what a small portion of the thyroid is left. More novocaine is injected just below the sternal notch for the drainage incision, so that the incisions are not interfered with. A cigarette drain is put in down alongside of the trachea.

We are now ready to restore the neck just as in the previous case. We must be careful not to sew this drain in, as the stitches are inserted in the muscles. We sewed one in with chromic catgut a little while ago, causing considerable trouble in removing it. Doctor Kinnie will now sew up the skin and platysma. This bulging drain

we do not care for; it will be removed in three days. She has slept practically throughout the entire operation.

Chronic Mastitis.—The next case is a woman, fifty years of age, married. Last fall she noticed blood coming from her right nipple. About one month ago she observed that the breast was enlarging, and it has been continuing to enlarge since. One week ago she consulted her physician, who referred her to us for operation. The remainder of her history is unimportant. General physical examination is negative except for the condition of the breast. Right breast presents a tumor about the size of a lime beneath the nipple and slightly to the right of it, irregular in outline, indurated, infiltrating surrounding tissues, and adherent to the skin. There are no glands to be felt in the axilla.

There are certain things about this tumor which to our minds make it appear malignant. First, the bloody discharge from the nipple is extremely suspicious of malignancy, although it may occur without malignancy. Next, it is adherent to the skin, which is also extremely suspicious; and, next, the tumor is indurated, irregular in outline and not sharply defined. Tumors adherent to the pectoral fascia or skin are very apt to be malignant. We will excise the tumor with the surrounding breast tissue, taking care not to cut into the tumor, and we will then send it to the pathological laboratory for immediate diagnosis.

Immediate pathological report on frozen section is chronic mastitis. Having excised the breast so that no breast tissue remains, the wound is closed with buried sutures of catgut and skin stitches of silkworm gut. Inasmuch as this is a benign tumor, the axilla will not be dissected and the pectoral muscles will not be removed.

This case is extremely interesting and instructive. It indicates that in a woman of this age the diagnosis between chronic mastitis and malignancy may not be easy; in fact, may be impossible, except by microscopical examination.

We have removed the entire breast in this case because of the danger of malignancy appearing in it later if any breast tissue be left behind. It is generally believed that malignant degeneration appears in breasts affected with this condition in from ten to fifty per cent. of the cases. In view of this fact, we must assume, par-

ticularly in a woman of this age, that this is a precancerous condition and remove it entirely.

If this had occurred in the breast of a young girl, for instance, we believe that we would be justified in removing only the affected part of the breast through a marginal incision and keeping the young lady under observation for a considerable period of time.

Although this woman did not have glands in her axilla, I wish to impress upon you that glands not uncommonly appear in the axilla with chronic mastitis, thus causing this condition to simulate that of carcinoma still further.

Report on frozen section (Fig. 3) from pathological department by Dr. Edgar S. Medlar:

The gross specimen consists of a breast in which is felt, on palpation, a firm nodule, about 2 Cm. in diameter, lying beneath the nipple. On section this area is found to be a cyst filled with blood. Near by is another similar area, which on section is found to be cystic. The wall of this cyst appears granular in one place. Scattered throughout the breast are other cysts, averaging about the size of a pea. Many of these are filled with blood. *Gross diagnosis:* Chronic mastitis.

Microscopical.—Section shows marked dilatation of glands, some of which are filled with blood; others are filled with finely granular material in which are deposited cholesterin crystals. There are a number of old hemorrhages undergoing organization. Aside from these organizing hemorrhages, marked areas of chronic inflammation are found. In some of the cysts filled with the homogeneous secretion are large numbers of endothelial leucocytes filled with fat and with a light-yellowish pigment. Arising from the wall of several of the cysts are papillary and solid growths of epithelium which partially or completely fill the lumen. In some places the tumor-cells are growing beneath the old epithelial lining, but in no place was there found any invasion of tissue beneath the basement membrane. Mitotic figures are numerous.

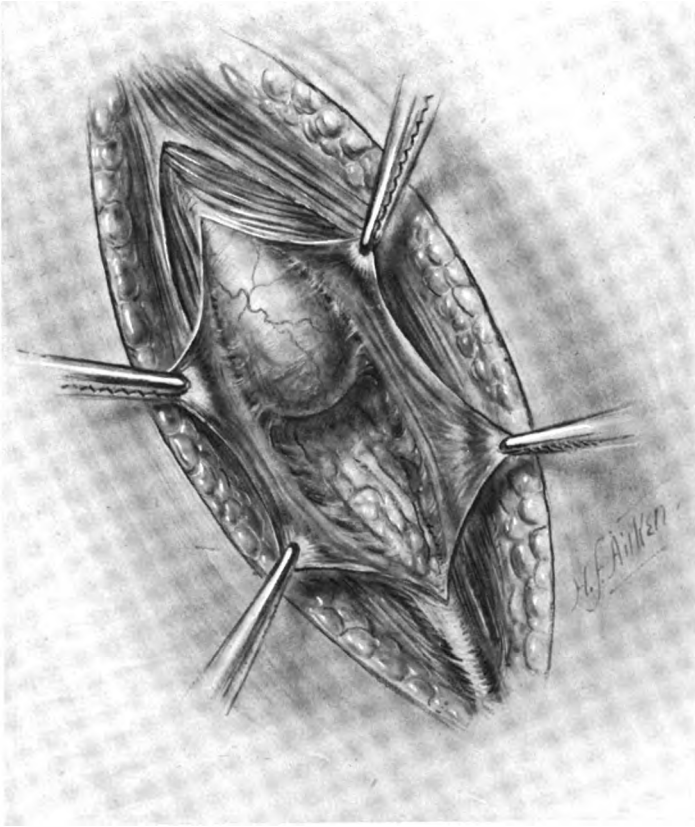
The case is one of marked chronic mastitis complicated by hemorrhage and cyst formation. The new growth of the glandular epithelium is to be regarded as mildly malignant or, perhaps better, as precancerous. In such cases radical operations are not necessary, but the breast should always be removed.

Inguinal Hernia.—The next case is a woman, fifty-five years of age, born in Russia, and married. For the last five years patient has noticed a swelling in the right groin, small at first and growing larger. When the patient is recumbent the swelling goes down; she can reduce it with her hands. It causes some discomfort on walking; otherwise is not painful. Remainder of physical examination is negative.

This woman has a right inguinal hernia on which we propose to do a herniotomy. There are two types of hernia operation; that is, for practical purposes there are but two types: (1) The Bassini operation, in which the cord is transplanted between the internal oblique and the external oblique; (2) the Andrews-Ferguson operation, in which the cord is left behind the internal oblique and conjoined tendon and emerges directly over Poupart's ligament near the spine of the pubes. There are very many other operations bearing their originators' names, but they are largely concerned with the method of dealing with the sac. It is our custom in indirect hernia or oblique hernia to use the Andrews-Ferguson operation; in doing this operation I wish to call your attention to a method of delivering the sac by separating the fibres of the cremaster, which we think makes the operation very much easier and simpler, with less danger to the cord and less danger of hæmatoma; it likewise makes suture of the internal oblique and the conjoined tendon to Poupart's very much easier, since veins, vas, and fat are kept within the cremaster and so from bulging up into the wound. We will demonstrate it to you on the case when she comes in.

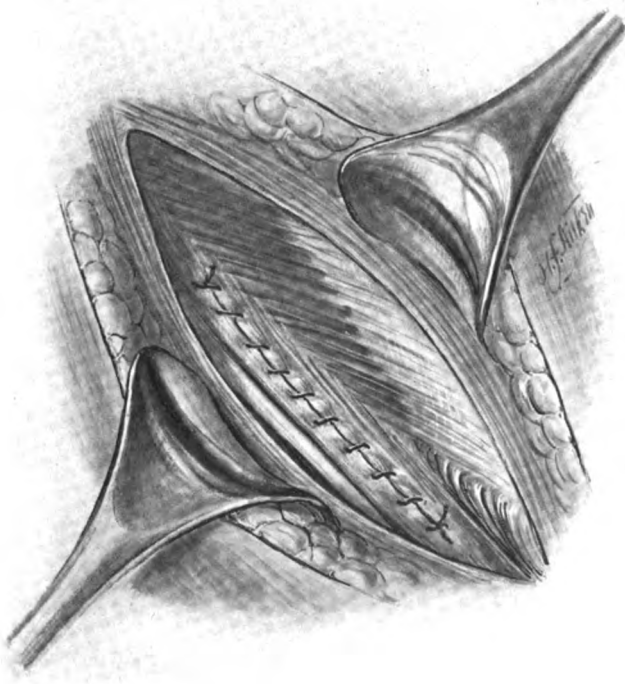
The incision for an inguinal hernia is about two fingers' length above Poupart's ligament and parallel to it. This incision is carried through fat and superficial fascia down to the aponeurosis of the external oblique. There are numerous superficial veins which must be clamped and tied. Here is the external oblique and the external inguinal canal, which is clearly marked by the intercolumnar fibres running transversely just above the ring. My finger, you can see, is now in the lax, external ring. There are the intercolumnar fibres which mark the ring. The external oblique is now cut up through the upper portion of the ring, care being taken not to cut off the ileo-inguinal nerve, which runs here on the edge of the internal oblique muscle. The external oblique is clamped and stripped back with a

FIG. 4



Showing the cremaster envelope held open and the sac bulging down beneath cremasteric fibres at the top.

FIG. 5



Showing restoration of the cremaster with the resulting clean canal facilitating the introduction of suture between conjoined tendon and Poupart's ligament.

piece of gauze, exposing the conjoined tendon. The lower portion of the external oblique, the reflection of which is Poupart's ligament, is wiped back, and Poupart's ligament then comes out plainly.

Now the point I wish to make in this operation is the method of delivering the sac from the cremaster muscle. Here is the lax cremaster muscle which surrounds the sac of the hernia. We pick up the cremaster and separate it in the direction of the fibres, grasp either side with a hæmostat, and the sac can be seen clearly at the upper part of the canal, grayish white in color and bulging down into the canal. Notice how the cremaster is left as an envelope surrounding the sac (Fig. 4). Notice that in oblique herniæ the vas deferens, which you can see plainly, is behind the sac. Now I think I can demonstrate very plainly to those of you who are close at hand the pale thin edge of the sac as it turns here. This is a thin, delicate sac; you can see the edge of it at this point. We will pick that up. Now you can see the pale color which characterizes the sac from the surrounding tissues. That is opened, its edges grasped, and the shiny peritoneal lining is plainly visible. Observe how easily it dissects out of this envelope of cremaster fibres. The assistant holds the adhesions on the stretcher and they are cut, care being taken that the cord is not cut. The cord is seen behind the sac. When the adhesions are cut, the sac is wiped out of the envelope of cremaster fibres, and is freed well up to its neck. First we investigate the contents of the sac and reduce any possible omentum or small intestine. It is then twisted on itself so that if a piece of omentum or intestine should get back into it, it will be reduced into the abdomen by the gradual twisting of the sac. This is then transfixed with a needle and ligated high up on its neck. The sac is now cut about half an inch from the ligature so that there is no danger of slipping. The cut end of the sac is carefully observed for possible bleeding. None is found, and it is dropped back into the abdomen.

We now have left this envelope of cremaster fibres which we will suture. Notice when they are sutured that the cord is within the cremaster and covered by these sutured fibres; also notice that the inguinal canal will be perfectly clean and free so that the sutures between the internal oblique, conjoined tendon, and Poupart's may be put in without difficulty (Fig. 5). Inasmuch as the suture of

the cremaster bears no strain, a continuous suture is quicker and easier to introduce.

You can see that the cremaster has been entirely restored, the cord is behind, and the inguinal canal is now free of bulging cord or veins, so that the kangaroo sutures may be put in without difficulty. Notice the course of the ileo-inguinal and ileohypogastric nerves. We shall be careful that these are not grasped in the sutures, because the ileo-inguinal has a portion which supplies sensation over the thigh, and if it is grasped hyperæsthesia is produced over this portion. The first stitch goes through the internal oblique and conjoined tendon, a good bite being taken of the ligament. Because of the liability to untying, kangaroo tendon should be cut so that about half an inch is left. (This Andrews-Ferguson operation is best used in oblique hernia, because in this type of hernias the conjoined tendon is not attenuated; in direct hernia the conjoined tendon is thin, atrophied, and does not allow of good approximation to Poupart's ligament at its lower portion.) Sutures are introduced until the inguinal canal is closed, except for a small triangular area beside the pubes, into which the tip of one's little finger may be barely introduced. This is the last stitch, and care must be used to see that a good bite of Poupart's ligament is obtained. It barely admits the tip of the little finger. The external oblique is united with plain catgut, and the skin with silkworm gut. These stay stitches go through skin, superficial fascia, and pick up the edge of the external oblique; they have two advantages: (1) They bring the bottom of the wound to the top and obliterate dead space, and (2) they take most of the strain in vomiting.

URETERAL CALCULI: SOME CARDINAL DIAGNOSTIC CONSIDERATIONS, TOGETHER WITH AN ANALYSIS OF TWO ILLUSTRATIVE CASES

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DIAGNOSIS is the point around which revolves the conduct of every surgical case. Especially is this true of ureteral calculi. This discussion has to do, not with an exhaustive technical consideration of differential diagnostic methods dealing with numerous urological conditions, but with pointing out certain diagnostic pitfalls which are ever present, wherein the symptoms due to the presence of a calculus are not easily recognized as of urological origin, and with emphasizing the need of more frequent urological investigation preceding much abdominal surgery.

Considerable speculation exists regarding the genesis of ureteral calculi. Keyes¹ dismisses the subject in his usual direct manner by saying, "All ureteral stones are kidney stones." If this be true, as it undoubtedly is, in at least a majority of the cases, we must inquire as to the conditions existing in the particular locality of the stone before its migration. Fowler,² after reviewing the literature on the etiology of renal calculi, is inclined to consider infection as a predisposing factor of prime importance, supplementing this by calling attention to the question of unnatural drainage of the lower calyces, even when the kidney is in normal position, and as being more pronounced when in prolapsus. Continuing, he describes in detail his operation for kidney anchorage to overcome this condition.

It is an undisputed fact that the presence of stones in any of the organic cavities is almost invariably preceded by the presence of some foreign substance, be it infection, blood-clot, or foreign body.

In view of this fact, the theory of infection seems entirely tenable. A possibility that has suggested itself to us—on account of findings in some of our recent cases—is that, following infection in a sacculated portion of the ureter, genesis of the calculus had occurred at this point.

THE SYMPTOMS AND DIAGNOSIS

During the early history of certain communities, justice was frequently administered first, and investigations conducted at leisure, if at all. If the histories of a large number of surgical cases were accepted at their face value, one might acquire the impression that the foregoing principle had been applied in certain cases of abdominal surgery. Let us see: Cabot,³ in a study of 153 cases of ureteral and kidney stone at the Massachusetts General Hospital, noted that 26 abdominal operations—other than for stone—had been done in efforts to relieve symptoms due to the presence of calculi.

In a review of 450 cases of stone operated on at the Mayo Clinic, Braasch⁴ found that 143 patients, or practically one-third, had had laparotomies previously performed elsewhere for relief of pain. The cases collected at the above clinics are drawn from a considerable territory and are, we think, a fair representation of general conditions.

From the foregoing only one conclusion can be drawn: certainly a thorough urologic examination could not have been made in these cases. Some sort of clue, from the urologic findings, must surely have directed one to a correct diagnosis in a much larger percentage of the cases.

Conditions Confounded with Calculus.—Let us first consider the several rôles assumed by the calculus that frequently mislead experienced operators into performing laparotomies which yield such disappointing results.

Pain is the instigating factor which impels the patient to seek the advice of a physician in all cases where calculus is diagnosed as some other condition. Pain is the false guide that leads the unwary operator into diagnostic pitfalls from which he is able, with ill grace, to extricate himself. Pain alone is not a dependable symptom, irrespective of its character or location. It may simulate calculus when no calculus is present, and, when due to calculus, may

present a picture of some condition entirely foreign to the reality. Pain, as a symptom of calculus, may be confined to the upper or lower abdomen, or both. Pain in the upper abdomen caused by ureteral calculus is due to distention within the kidney from back pressure by reason of ureteral occlusion due to lodgement of the calculus. When in the lower abdomen the pain is due to local inflammatory processes taking place in the ureter or surrounding tissues, following the lodgement of the calculus. This pain, in many instances, may not be confined to the region of the ureter, but may be referred to any portion of the lower abdomen.

Squier,⁵ in discussing renal pain, states that the four main pathologic conditions apt to be confounded with renal lesions by reason of this symptom are: (1) coincident disease of the gall-bladder and ducts, (2) gastroduodenal ulcer syndrome, (3) appendicitis, and (4) affections of the large intestine. Pain as a symptom due to the above is largely confined to the upper abdomen.

Considering pathologic conditions in the lower abdomen likely to be confounded with ureteral calculus, we would append the above by including (1) affections of the Fallopian tubes and ovaries in the female, (2) affections of the vas deferens and seminal vesicles in the male, and (3) vesical calculus.

It is not those cases in which pain, due to ureteral or renal lesions, is typical or classical that we wish to consider, but those cases in which this symptom is positively atypical, and in which from this symptom alone a correct diagnosis can seldom be made.

A calculus may become lodged and produce occlusion of the ureter and be responsible for symptoms commonly encountered in any of the previously enumerated pathologic conditions wherein pain is referred to the upper abdomen; namely, diseases of the gall-bladder and ducts, gastric or duodenal ulcer, appendicitis, and affections of the large intestine—mostly obstruction. General abdominal pain, nausea, and vomiting may be present—and, apparently, the only symptoms present during the onset of either of the above conditions or in renal colic.

Many writers on general diagnosis, in discussing this situation, contend that a simple microscopical urinary analysis shows blood or pus, or both, with ureteral calculus. Undoubtedly this is true in the usual run of cases, but it is the unusual cases which we wish to

consider, and it is with the unusual cases that most mistakes are made. We can hardly conceive of a competent surgeon opening an abdomen in any of the above conditions without some sort of urinalysis having been previously made; but the thing we wish to point out in this connection is that a negative urinalysis does not exclude calculus in the unusual cases. Cabot,⁶ in reviewing 150 cases of proved calculus, found the urine normal in 14 per cent. on several repeated examinations.

What, then, is the answer? Only one logical conclusion. When dealing with pathologic conditions, causing abdominal pain, unless stone in the ureter can be ruled out by some diagnostic factor directly in favor of the other condition, a complete urologic examination should be made. This we shall discuss later.

In the conditions previously classified as existing in the lower abdomen—namely, ovaritis and salpingitis, seminal vesiculitis and vasitis, and vesical calculus—the pain is apt to be more local in character and accompanied by local tenderness.

For the purpose of comparison, let us briefly consider each of the foregoing conditions as against ureteral calculus, presupposing the absence of the classical symptoms of ureteral calculus; namely, pain and tenderness over the ureter, with pain radiating to the groin and inner thigh, scrotum, or labia; blood or pus in the urine.

1. Pain in the upper right abdominal quadrant, radiating to the back, intermittent and excruciating, may be present in either ureteral calculus or *gall-stone colic*. Toxic jaundice may exist with calculus, simulating the obstructive jaundice following occlusion of the common bile-duct. Evidence indicative of obstruction of the cystic or hepatic duct should be: rising temperature, high leucocyte count, local tenderness, and possible demonstrations of shadows in gall-bladder region by X-ray. Without these, or with blood or pus in the urine, a urologic examination to exclude calculus should always be made before exposure of the gall-bladder.

2. Pain caused by calculus lodged in the ureter just outside the kidney pelvis may give rise to pain simulating *gastric* or *duodenal ulcer*. This pain may be entirely confined to the epigastrium; it may be constant or intermittent in character. In ulcer, one should find local tenderness and gastroduodenal changes as demonstrated by stomach analysis and the X-ray findings with ingestion of bismuth

or barium. Again, the so-called "hunger pain" existing with ulcer should be followed by relief after food ingestion. When likely to be confounded, ulcer or calculus is usually of the so-called "chronic type," and ample time exists for urologic examinations in the doubtful cases.

3. *Appendicitis* is, probably, the diagnosis most often made when symptoms due to ureteral calculus receive a misinterpretation. The onset of either may be heralded by diffuse abdominal pain, vomiting, and, later, local tenderness over McBurney's point, especially when a calculus is lodged in the lower right ureter. Rectal palpation may elicit tenderness in either condition when the appendix dips down into the pelvis. Hæmaturia may be found in either. The principal diagnostic factor, as between appendicitis and calculus in the early stages, is the presence of an ascending temperature and leucocytosis in the former and an almost constant absence of these in the latter. In the chronic conditions a decision must be made from the evidence furnished by complete digestive and urologic findings.

4. *Intestinal obstruction* is the rôle sometimes assumed by ureteral calculus. Although rare, this mistake in diagnosis occurs. In some cases of calculus the diffuse abdominal pain and vomiting, when accompanied by abdominal distention, are suggestive of obstruction. Intestinal obstruction, however, as a usual thing, if observed closely, soon shows symptoms peculiar to obstruction. If a diagnosis can not be made with certainty, the urologist should be required to rule out ureteral calculus before operation.

In the lower abdomen, affections of the ovaries, Fallopian tubes, seminal vesicles, and vas deferens are interesting, in this connection, chiefly because a calculus may be present in the lower ureter coincidentally with these conditions. Frequency and tenesmus with slight hæmaturia or pyuria may accompany either condition.

Symptoms due to inflammatory changes in these pelvic structures may overshadow symptoms due to a calculus which may entirely escape the attention of the attending surgeon. This possibility should be kept in mind and urologic examinations made in doubtful cases.

Frequency of urination, with attendant vesical distress, commonly seen in vesical calculus, is, at times, the outstanding symptom in cases of ureteral calculi. Records show that not a few normal bladders have been opened for the purpose of removing a vesical calculus,

much to the chagrin of the operator. The most elemental sort of cystoscopy would have made these monumental blunders impossible.

TECHNIC OF UROLOGIC EXAMINATION TO DETERMINE PRESENCE OF URETERAL CALCULI

Not many years have passed since operations were performed for the removal of ureteral calculi, where the diagnosis was based on clinical observation alone. Naturally, this practice furnished many unpleasant surprises for the operator. To-day the competent surgeon demands more substantial evidence. Conclusive evidence of calculus—retained calculus—can be obtained from just two sources—the X-ray and the cystoscope. In a certain percentage of the cases a combination of both is imperative, as the evidence derived from either, alone, is subject to error.

Cabot,⁷ in a graphic discussion of "Errors in Diagnosis of Renal Calculus," has aptly said: "To the trained urologist the most interesting man-traps are set for him by his association with the imaginative radiologist. Of the cases coming to us with symptoms referable to the kidney, and more or less typical of renal colic, a considerable number are reported as showing shadows in the course of the urinary tract which, according to the temper of our expert brother, are said to be probably calculi or probably not calculi. So far as I can determine, the probability depends largely upon whether he has recently been mistaken on the positive or the negative side. A burnt child fears the fire, but, as the radiologist is situated between two fires, he can take refuge in either, to the confusion of the urologist." This is equally true in cases of ureteral calculi. Without the cystoscopist, the radiologist, in dealing with cases of ureteral calculi, is "a ship without a rudder." Phleboliths, calcified tuberculous glands, and other shadow-casting agents may be located along the course of the ureter. The differential diagnosis rests with the cystoscopist. Conclusions must be drawn from the following:

1. Intravesical cystoscopic observation.
2. Ureteral exploration with wax-tipped catheters.
3. Radiographs with shadowgraph catheters in the ureters.
4. Ureterograms with the ureters filled with thorium, collargol, or other shadow-casting agents.
5. Urinalysis.

FIG. 1



FIG. 2



1. *Intravesical Cystoscopic Observation*, in this connection, is of value for two purposes. It reveals whether or not vesical calculi are present. It shows the condition of the ureteric orifices and whether or not a calculus is lodged therein.

2. *Ureteral exploration* with wax-tipped catheters, when properly done, shows the presence of stone—when stone is present—in a large percentage of the cases. The catheters should be passed into the bladder ahead of the cystoscope to avoid scratching. The wax tip, in passing over the rough surface of the calculus, receives certain scratches, wherein lies its diagnostic value. In the use of this method, the possibility must be kept in mind of a calculus being so situated or with a surface of such smoothness that no impression on the wax results. A positive finding is diagnostic for, but a negative finding is not diagnostic against, calculi.

3. *Radiography* is indispensable not only in demonstrating the presence of stone, but its location, with exactness. The radiologist, however, must be guided and assisted in his findings by the urologist. Competent observers declare that radiography alone fails to demonstrate stone in about 15 per cent. of the cases. Radiography alone is many times misleading, in that shadow-casting agents along the course of the ureter may be mistaken for calculi. This error may be avoided in practically all cases by the use of shadowgraph catheters. The shadowgraph catheter shows on the X-ray plate the exact relation of the shadow-casting agent to the ureter. Figs. 1 and 2 demonstrate this necessity. In this case the clinical symptoms suggested very strongly the presence of calculus, and Fig. 1 would seem to substantiate the evidence. Fig. 2 shows the error.

A few cases are on record where calcified tuberculous glands situated just outside the ureter caused a partial closure of the lumen, and were not diagnosed as such until operation, although radiograph catheters were used. However, their removal provided relief from pressure and perhaps later calculus formation.

4. *Ureterograms*.—It is quite generally agreed that the calcium content of calculi is the shadow-casting element. It is very probable that it is in the cases where this element is mostly or entirely absent, such as uric acid calculi, etc., that the most errors in radiography occur. It is in these cases that intra-ureteral instillations of thorium, collargol, etc., provide a means of bringing more of these cases to

light. This method demonstrates the exact contour of the ureter; shows constrictions and sacculations, suggesting the possible location of the suspected calculus. We have discovered calculi in this way (large uric acid calculi, which otherwise we were unable to demonstrate) by reason of the non-shadow-casting calculus being surrounded by the thorium, and showing less density in the plate shadow. Radiographs made soon after drainage of the shadow-casting agent from the ureters sometimes demonstrate the presence of small calculi otherwise not demonstrable, by reason of some of the agent remaining on the surface of the calculus, thus casting a slight shadow. In a small percentage of cases it is impossible to enter the ureter with any sort of catheter. The impossibility of the above-described technic is therefore quite obvious. In these cases the only remaining thing to do is to incise or dilate the orifice through the operating cystoscope sufficiently to admit ureteral catheters.

5. *Urinalysis*.—Urine obtained from each ureter separately should be examined. While not a constant symptom, blood or pus from either ureter, when present, is evidence suggestive of calculi. To avoid a certain percentage of error in radiographic technic, repeated examinations should be made unless the early findings prove satisfactory. Another point worthy of consideration is that a calculus may change position over night; for this reason it is the part of wisdom to have a radiograph made, for the purpose of comparison, just prior to entering the operating room.

Pain may be referred from the affected to the unaffected side. Owre,⁸ in this connection, has pointed out the advisability of the examination of both sides in these cases. Where doubt exists as to the side involved, ureteral catheterization will, of course, provide a clue when hemorrhage or pus is present. However, in regard to blood, one must keep in mind the possibility of trauma in passing the catheters. To be of diagnostic value, the hemorrhage should be of more volume than a few scattered red cells in the urine.

SUMMARY

1. Ureteral calculus is, in many instances, responsible for symptoms which may closely resemble those due to several other abdominal conditions and which, if not carefully investigated, frequently lead to a diagnosis other than calculus. Many unnecessary operations

are performed in consequence, resulting obviously in no change in the urologic condition.

2. Pain, in calculus, is often atypical and may simulate the pain caused by a number of other abdominal pathologic conditions, and when taken alone is not a reliable diagnostic agent.

3. The urine is persistently negative in many cases of ureteral calculus, but in combination with other data may provide important diagnostic evidence.

4. Competent observers state that as many as one-third of the cases of calculus seen by them had been previously operated upon for conditions other than calculus, when the symptoms which induced the surgeon to operate were due to calculus.

5. We consider this percentage of mistaken diagnosis unnecessarily high, and think that, with a careful urologic examination in the doubtful cases, this percentage could be greatly lowered.

6. Diagnostic evidence, to be conclusive, in ureteral calculus, must be derived from a combination of the following: Clinical observation; urinalysis, from ureteral catheterization; cystoscopic observation; exploration with wax-tipped catheters; and radiography done in conjunction with shadowgraph catheters and with intra-ureteral instillations of thorium, collargol, or other shadow-casting agents.

7. The evidence, when taken alone from any one of the above sources, is apt to be misleading; but in cases where calculus is present some one or more of the methods given is almost certain to provide evidence leading to a correct diagnosis.

CASE I (seen and operated upon in association with Dr. J. N. Jackson).—Mrs. D., age forty-nine. Married. Two children. History unimportant until last childbirth, which occurred 23 years ago, and which was complicated by puerperal septicæmia. Four years later right tube and ovary were removed, following a diagnosis of salpingitis. Beginning soon after the operation, and continuing up until the present time, she has had intermittent attacks of pain in lower right abdomen. Six years ago she had an unusually severe attack which was accompanied by nausea and vomiting. Since that time she has had almost constant distress in the right lower abdomen. Beginning several years ago, she also had intermittent attacks of rather indefinite pain involving the upper and lower abdomen on the

left side. Recently she has been troubled with frequent and painful urination.

Examination.—Genitalia negative. Abdominal tenderness can be elicited over right iliac quadrant only. Urine (from bladder) negative, except considerable pus and colon bacilli.

Cystoscopy shows bladder mucosa somewhat reddened. Right ureteric orifice normal. Left ureteric orifice enlarged and gaping.

Radiography.—Fig. 3 shows probable calculi in both ureters.

Ureteral Catheterization.—Catheter passed without difficulty into right ureter, the urine from which was normal. In attempting to catheterize the left ureter, an obstruction was met with about 7 Cm. above the bladder. Unable to pass catheter beyond this point. No urine obtainable.

Radiography with Shadowgraph Catheters.—Fig. 4 shows the shadow-casting agents along the course of the right ureter to be situated outside of ureter. At the end of catheter in left ureter is shown calculus, probably occluding ureter. Probable calculus about 4 Cm. above first calculus.

Pyelography.—Fig. 5 shows probable stone in left kidney.

First Operation, October 14 1916.—Abdomen opened under ether anaesthesia. A Meckel's diverticulum situated about 25 Cm. from the ileocaecal valve was removed. The tip of the diverticulum was tightly adherent to the anterior abdominal wall in the region of the anterior superior iliac spine. Numerous adhesions of the omentum and ileum to the broad ligament on the right side were removed. Normal appendix removed. On the left side a calculus about $\frac{1}{2}$ Cm. in width and 1 Cm. in length was removed from the left ureter. A ureteral catheter was inserted into the ureter for drainage. No attempt was made to remove the other ureteral calculus nor the probable calculus in the left kidney. Patient made an uneventful recovery.

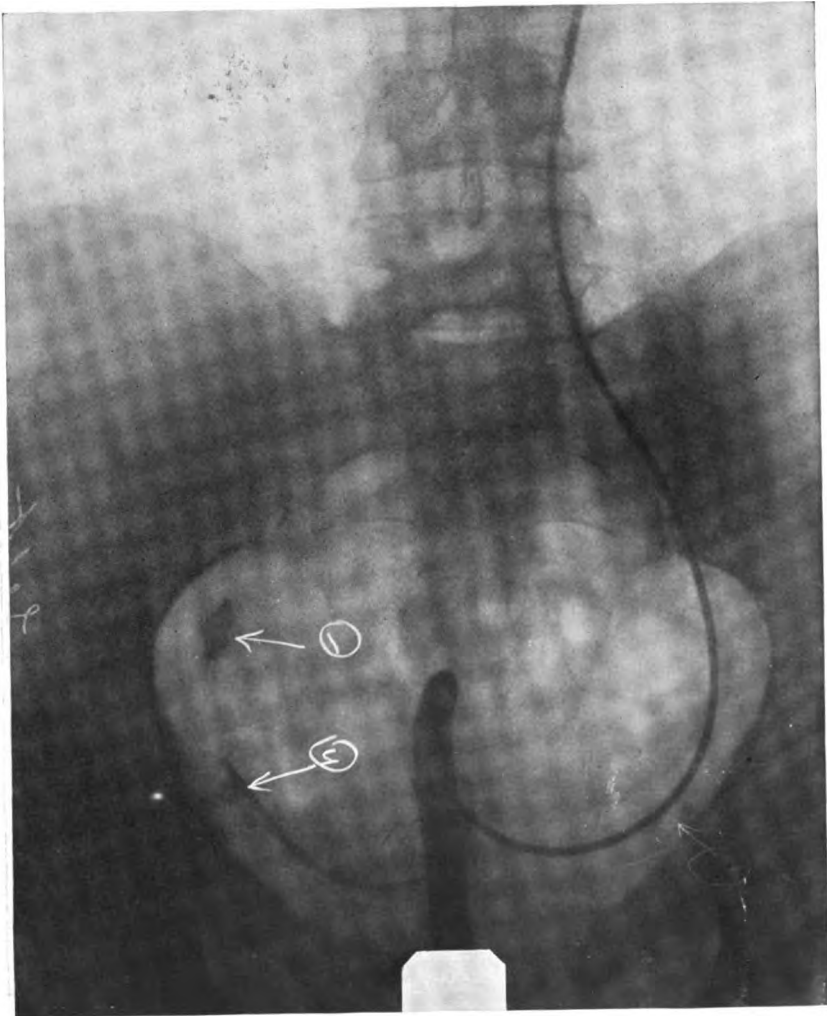
Second Operation, March 26, 1917.—Left kidney and ureter exposed by modified lumbar incision under ether anaesthesia. Calculus about 1 Cm. in length and $\frac{1}{2}$ Cm. in width was removed from left ureter, about 10 Cm. above bladder. On exposure the left kidney was found to be about 5 Cm. in length and $2\frac{1}{2}$ Cm. in width. The pelvis contained a calculus of about the size removed from the ureter. It was quite evident that no function had existed for some

FIG. 3



Case I

FIG. 4



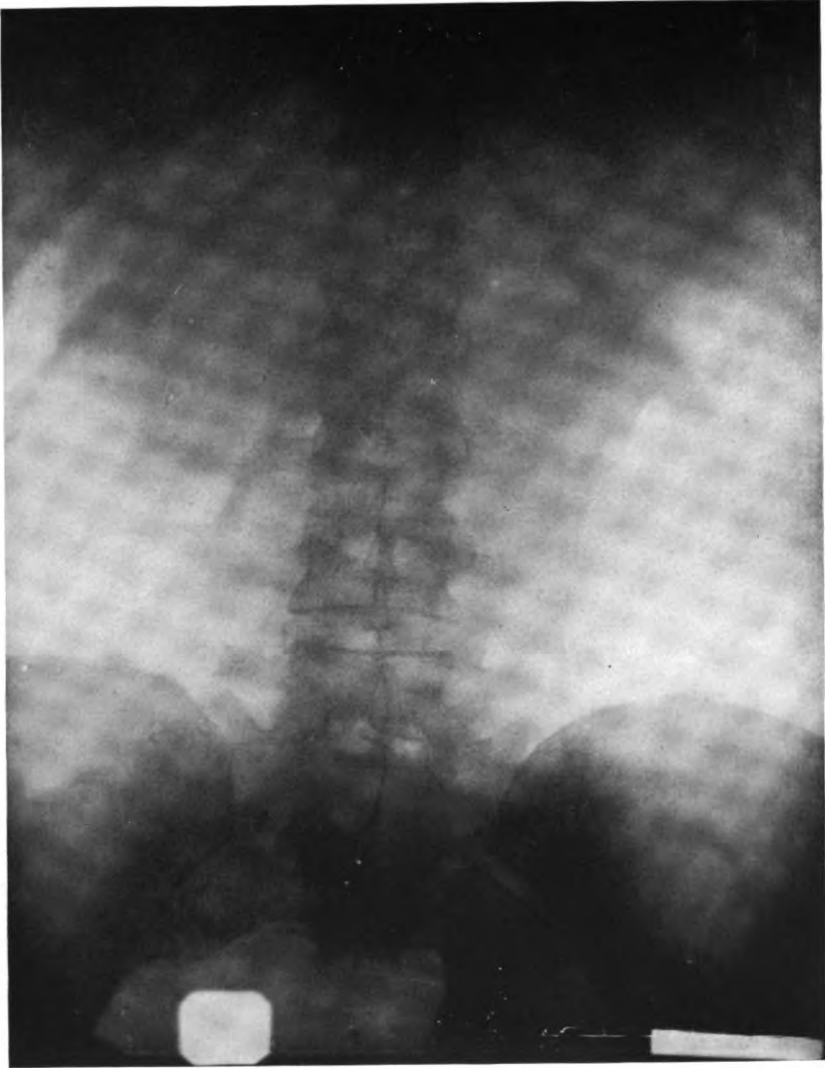
Case I

FIG. 5



Case I

FIG. 6



Case II

time, probably due to a complete occlusion of the left ureter. For this reason a nephrectomy was done. Convalescence was uneventful.

Interest attaches to this case for the following reasons:

1. Probable kidney infection at time of puerperal septicæmia, followed later by calculus formation.

2. The absence of any definite clinical symptoms of calculus on the left side.

3. Probability of the severe attacks of pain on the right side being due in part to presence of calculus on left side. The pain referred.

4. Destruction of left kidney due to complete ureteral obstruction, which, had it been discovered at an early date and properly treated, would have precluded the possibility of kidney destruction.

CASE II (referred by Dr. N. O. Harrelson).—Mrs. S., age thirty-eight. No children. Family history unimportant. Previous history: Received injury in right lumbar region when about six years of age. The immediate effect of the injury was such as to necessitate her remaining in bed several weeks. A few months later she began to experience intermittent attacks of pain in the right abdomen. These attacks, accompanied at times by frequency of urination, have appeared at intervals of two to five weeks, and have persisted up until the present time, notwithstanding two operative attempts to relieve the condition.

Twelve years ago this patient had an unusually severe attack of pain in the right abdomen, during which the pain was referred to the right inguinal region. A diagnosis of "floating kidney" was made, followed by exposure of the kidney and subsequent drainage for about six weeks. The history here suggests a likely infection or, at least, pus condition of the kidney at that time.

Following this operation, this patient experienced relief from her pain for a period of several months. Subsequently the attacks appeared as before.

About six years ago a laparotomy was performed, in which the appendix, right tube, and ovary were removed. The patient again was relieved of her pain, this time for a considerable number of months. Again the attacks of pain appeared and have continued intermittently up until the present time.

Examination.—Abdominal tenderness over right side only, be-

ginning at about the level of the umbilicus and extending into pelvis.

Urinalysis.—Bladder urine contained large amount of pus, considerable colon bacilli, and some red blood-cells.

Cystoscopy.—Bladder mucosa slightly red and congested. Left ureteral orifice normal in appearance. Right ureteral orifice inflamed and gaping; pus exuding.

Ureteral Catheterization.—Left ureter, catheter passed freely. Urine normal. Right ureter, catheter met with impassable obstruction about 6 Cm. above ureteric orifice. Small amount of urine was obtainable, which contained pus, colon bacilli, and red blood-cells.

Radiography.—Fig. 6 shows what appears to be a mammoth ureteral calculus, situated about 6 Cm. above right ureteric orifice.

Pyelo-ureterogram.—Fig. 7 shows a much dilated ureter above the supposed calculus, also some attendant dilatation of kidney pelvis.

Operation, February 22, 1917.—Under ether anaesthesia the right lower ureter was exposed, extraperitoneal, through a modified lumbar incision. A calculus, measuring about $7\frac{1}{2}$ Cm. in length and 3 Cm. in width and weighing 44 Gms., was removed (Fig. 8). The ureter was enormously dilated and thickened above the calculus, and contained a large amount of bloody fluid, which was under considerable tension and escaped with a rush as the ureter was incised.

As fairly good function existed, it was thought expedient not to institute operative interference with the kidney, but to establish drainage per ureter and await results. Accordingly, a large drainage-tube was placed in the wound, reaching to the incision in the ureter, which was not closed.

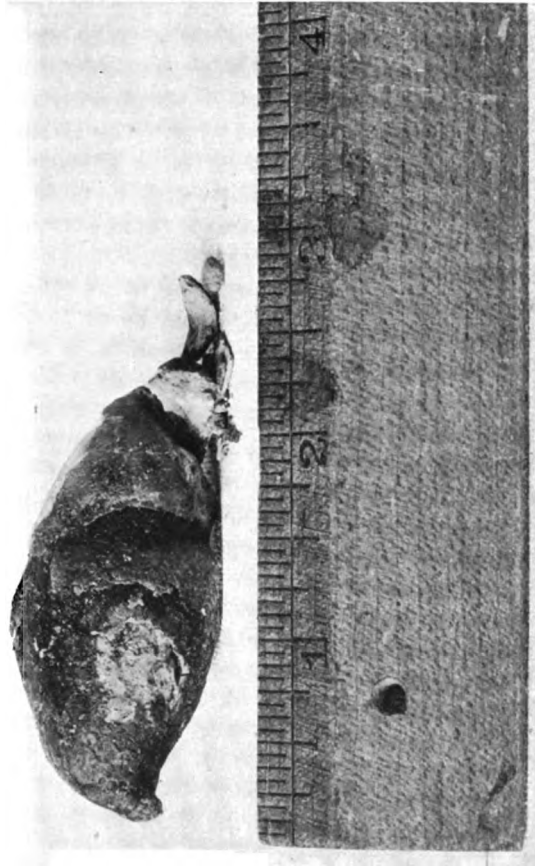
The patient had a rather stormy convalescence, complicated by ether pneumonia and toxic jaundice. During the first few days following operation, very little urine appeared from the affected side, either through the wound or the ureter into the bladder. After about the first week, however, the urine drained freely from the wound, and thereafter for about six weeks, when the wound closed. At the time of closure the urine was clear. This patient made an uneventful recovery from this point. Urine still contains some pus, but good kidney function exists. Needless to say, she is relieved of the attacks of pain, has gained considerable weight, and feels entirely well.

FIG. 7



Case II

FIG. 8



**Photograph of stone removed from ureter in Case II. Actual size.
Weight, 44 grammes.**

The principal points of interest in this case are:

1. The early history wherein injury was very likely done to the right kidney or ureter. From a speculative standpoint, the formation of the calculus may have occurred in the kidney, later becoming lodged in the ureter; or the calculus may have had its beginning primarily at the site of injury in the ureter, if such injury occurred.

2. The immensity of the calculus leads us to think that its formation must necessarily have covered a period of quite a number of years, and likely during the entire time in which the colicky pain existed in the right abdomen.

3. Two surgeons of recognized ability performed two separate operations and, to say the least, discovered no calculus.

4. Kidney function was greatly impaired and almost lost as a result of the delay in the discovery of the calculus.

5. Most any sort of radiography or ureteral catheterization would have provided the necessary clue for a correct diagnosis.

6. The seriousness of the case when finally brought to operation and the difficulty of removing a calculus of such mammoth proportions.

7. The gratifying results obtained, including restoration of kidney function.

REFERENCES

- ¹ KEYES: "Diseases of the Genito-urinary Organs," 1910.
- ² L. S. FOWLER: *Trans. Sec. G.-U. Diseases, A. M. A.*, 1913, p. 55.
- ³ CABOT: *Surg., Gyn. and Obstet.*, vol. xxi, No. 4, p. 403.
- ⁴ BRAASCH: *Ibid.*, vol. xxiv, No. 1, p. 11.
- ⁵ SQUIER: *Ibid.*, vol. xxi, No. 4, p. 413.
- ⁶ CABOT: *Ibid.*, vol. xxi, No. 4, p. 404.
- ⁷ CABOT: *Ibid.*, vol. xxi, No. 4, p. 405.
- ⁸ OWRE: *Med. Lancet*, March 1, 1917.

TWO LECTURES ON INJURIES TO THE CRANIUM AND BRAIN IN WARFARE

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LECTURE I.

GENTLEMEN: In what I am to say in these lectures on injuries to the skull and brain in warfare it is to be understood that I shall only refer to the conditions met with and the treatment to be applied in ambulance practice at the front. I would also add that the case-histories which I shall give are those reported from time to time from various ambulances, and represent, I think, what may be considered typical, and therefore I offer them as illustrative instances of daily practice.

Before entering into the subject proper I would like to briefly refer to the results obtained so far, by giving you a few statistical figures from the practice of several surgeons. When operated on at once, wounds of the skull may be recovered from, but their prognosis is nevertheless very different, according to the depth of the wound. When the dura mater has been perforated, when the cerebral lesions are extensive, death is the usual outcome. This is the case almost invariably for bullets which have gone directly through the head and for injuries resulting from an exploding shell, the pieces of which deeply penetrate and cause vast cerebral lesions.

When the missile does not penetrate deeply or when the dura has been opened, the patient often appears to be doing well for the first two or three weeks following the receipt of the injury, when suddenly the temperature goes up and the subject dies in a few days from diffuse meningo-encephalitis. However, in some instances, when the brain lesions are not too extensive and the patient escapes from a meningeal infection, he may recover even if the dura has been perforated. When the dura is intact, recovery is the rule, and nearly all cases trephined in these conditions get well.

By recovery I, of course, mean *immediate* recovery, because great reserve must be made in the ultimate prognosis of these cases. Not infrequently after a long lapse of time from the receipt of the injury, sometimes as much as several years, an abscess of the brain may develop which, if not recognized early in its evolution and operated on in time, causes death.

On the other hand, meningeal adhesions are prone to develop at the point of cranial injury, and it is perhaps to them that sudden death occurring a long time after recovery from the wound is due, when no other factor is revealed at autopsy.

Finally, you must not overlook the fact that the destructive lesions resulting from *cerebral contusion* are always permanent and create incurable infirmities, such as hemiplegia, monoplegia, and, frequently, cecity, while the intellectual faculties may be clouded and the memory weak. Such sequelæ are important when making the prognosis.

Now as to the immediate results of treatment in ambulance practice. Koechlin has recorded 42 cases of trephining for head injuries with cranial lesions, and of these 13 were from rifle bullets and 29 from bursting shells or bombs. Of the 13 bullet wounds, six were through-and-through, with five deaths, a mortality of 83.3 per cent. Seven cases were wounds where the missile hit tangentially, and among these there was one instance of fracture of the internal table without injury to the dura. This patient recovered. The six other cases presented a tear in the dura, and of these four died, a mortality of 66 per cent. The total mortality of bullet wounds was consequently 69.2 per cent.

Of the 29 injuries from exploded shells, in 15 cases the dura remained intact and all the patients recovered. In two cases the dura offered superficial contusion and both patients recovered. Finally, in the 12 remaining cases there was perforation of the dura with nine deaths, a mortality of 75 per cent. Thus the death-rate of wounds from exploding shells was 31 per cent., which is distinctly lower than that of bullet wounds, while the total mortality of the 42 cases was 18, or 40.4 per cent.

Lapointe has published the statistics of 127 cases, seven of which were through-and-through bullet wounds, with six deaths. There were 25 cases of penetrating wounds, with 14 deaths. In 48 wounds *without penetration*, in which the dura and brain were injured, there

were 27 deaths. There were 46 wounds without penetration of the projectile and where the dura was not injured. These included nine instances of depressed fracture of both tables, with three deaths.

There were 25 cases in which loss of substance was limited to the external table, with shattering of the internal table, with three deaths, while in nine cases of fissure of the external table all the patients recovered. In three cases of fracture of the internal table without lesions to the external table, one patient died.

Gross reports 35 cases of head injury with a total mortality of 41.3 per cent., while out of a total of 82 cases reported by Froehlich there were 20 deaths, or a mortality of 25 per cent. You see, gentlemen, how statistics vary from one observer to another.

Auvray reports that out of a total of 25 trephinations, nine were for slight lesions of the external table, with fracture of the internal table without injury to the dura, and all recovered. In 16 other cases there were injuries to the meninges and brain, only three patients recovered!

Coutland and Bellot have published statistics of 42 trephinations, among which there were 16 simple wounds without penetration of the projectile, with five deaths; five depressed fractures without injury to the dura, all recovered; and eight transversal wounds of the brain with four deaths, a total mortality of 33.7 per cent.

I know that figures are dry to listen to, and the only reason that I have bored you with these is to show that statistics of head injuries in warfare agree, and that the general conclusion to which one may logically come is that head injuries from rifle bullets are more serious than those resulting from exploding shells, because the former cause more extensive destruction. But what is still more important is the fact that when the dura remains intact recovery is the rule when they have been dealt with surgically in time, while death is most likely in cases where this membrane has been perforated.

Bullets produce variable cranial lesions, according to the angle at which they hit and also according to the distance which they have travelled before striking. When they hit the skull perpendicularly to its surface they usually go through, unless it be a spent bullet, but the resulting wounds will depend upon the velocity of the missile.

When fired from a short distance (50 to 100 yards), and therefore travelling at high velocity, the bullet will produce bursting of

the head with multiple splinters of bone, but, as such lesions cause instant death, they are devoid of surgical interest.

At a greater distance, 1200 to 1500 yards, the resulting lesions, although serious, are less extensive. The entrance aperture is round, clean cut and usually small—at least in the outer table and scalp. In the inner table, on the contrary, it is larger, irregular, and gives rise to bone splinters of various sizes, but they are generally triangular and assume the shape of a pyramid, with the apex pointing toward the brain, into which it often plunges.

The bullet produces similar lesions at its point of exit, but in this case the clean-cut aperture is in the inner table, while the outer table is splintered. The fissuring occasionally produces large splinters, as, for example, in the following case, in which the bullet perforated the skull above the eyebrow on the left and made its exit in the right frontotemporal region, producing multiple splinters in the frontal bone.

This patient was brought to the ambulance in a state of coma with a diagnosis of bullet wound of the skull. A wound was seen above the left eyebrow, which was the entrance wound, the exit aperture being in the right temporal region. By palpation one could feel multiple fractures extending between the two wounds, and, as the case was considered inoperable, an aseptic dressing was applied.

On the following days the patient's condition improved and the coma was less complete. Temperature nearly normal; free suppuration of the wounds. On the ninth day the temperature went up to 104° F., and the suppuration was still very plentiful.

Under chloroform, a long incision in the scalp revealed a comminuted fracture, with large splinters, of the frontal bone. The detached splinters of the frontal bone were removed and the wound rapidly cleansed. Rubber drain.

The next day suppuration was more abundant, the temperature was lower, but the coma was still deep. Three days later the temperature went up again, and the patient died in 48 hours with a temperature of 105.4° F.

In another case the bullet entered the right temporal region and made its exit in the occipital region, causing a large loss of bone and issue of brain matter. The patient was comatose, dying four hours later.

When the bullet is nearly spent it will probably remain in the brain. In a case where the patient was hit while lying down, the bullet entered the top of the skull, but its exit aperture could not be found. The trajectory appeared to be directed towards the basilar apophysis of the occipital. When brought to the ambulance, a small round wound was found on the cranial apex, through which some cerebral substance and clot issued. The patient was comatose. Lumbar puncture gave issue to very bloody fluid under some hypertension.

Under general narcosis an incision was made and the aperture in the bone enlarged with a Doyen drill. Some splinters from the internal table which pointed toward the base were removed. The trajectory of the bullet appeared to extend vertically from above downward. A wick was inserted and a flat dressing applied. After the patient had been returned to bed there was a severe hemorrhage. Death took place the following day in coma.

When the bullet hits the skull in a tangential direction it produces a more or less deep gutter in the bone which may involve both tables, thus producing a loss of substance of variable length, but the breadth does not exceed the diameter of the missile. As illustrating this, the two following cases will do.

The first was a penetrating bullet wound in the left parietal region. The patient walked from the automobile to the ambulance, saying that he was only slightly hurt and simply complained of a little headache and some difficulty in moving the right arm. While having his wounds dressed a wound of the scalp about ten centimetres long was found at the upper part of the left parietal region, with loss of bone and issue of brain substance.

The next day there was paresis of the right side. Temperature nearly normal. On the following evening the patient had an epileptiform seizure, localized to the right side of the body, while the next day the seizures became more numerous and the patient semi-comatose.

Next morning, under general narcosis, a scalp incision was made and the aperture in the skull was enlarged and evened off. Some splinters from the internal table, which penetrated the brain quite deeply, were removed. Flat dressings.

After the operation the epileptoid seizures recurred, but became fewer and finally disappeared. The patient's general condition im-

proved so that he was transferred to a base hospital six days later in very fair condition.

The patient was heard from ten months later, when he wrote that the epileptoid seizures had not recurred, but that he still had a marked decrease in the strength of his right arm and that he was often dizzy when he bent his head downward.

The second case is similar. There was a bullet wound striking at a tangent, producing a wound about ten centimetres long by one in breadth in the right parietal region. The dura was opened and gave issue to brain substance. There was a left hemiplegia without facial paralysis. The patient replied to questions with difficulty, was extremely agitated, but had not lost consciousness. Temperature normal.

Three days later, under general narcosis, the wound in the skull was evened and some splinters from the internal table, which entered the brain, were removed. Flat dressings.

The days following the patient was very restless, so much so that he had to be tied to the bed. Left hemiplegia still present.

Three days after the operation there was a small cerebral hernia, which slowly regressed. Eleven days after the operation the patient was transferred to a base hospital, where he died some weeks later.

In other cases the track of the bullet may involve only the external table, in which it makes a more or less deep gutter, sometimes only a simple abrasion, but such lesions are rarely single, and in the majority of cases the internal table is fractured and offers far more extensive damage than the external table.

The splintering of the internal table varies in extent and the splinters themselves vary in shape and size, but usually they extend much farther than those of the external table. For example, a soldier presented two wounds in the scalp; the entrance aperture was in the right parietal region, that of exit was in the left parietal region. Between the two the external table was depressed.

In the following case there was a small depression of the external table in the right parietal region, but the splinters of the fractured internal table penetrated the cerebral substance. The entrance and exit apertures were quite far apart, and it was supposed that the skull was injured. The patient was very dull and presented a left hemiplegia without facial paralysis.

Under general narcosis a scalp incision was made, when a depression of the vault was revealed. The opening in the skull was enlarged and splinters from the internal table, projecting into the brain, were removed. One of them was four centimetres long and one centimetre wide. A wick was inserted and flat dressing applied.

The patient progressed well, but two days later he developed seizures of Jacksonian epilepsy, limited to the left side. The seizures occurred about four times a day, but became fewer, so that the patient was transferred to a base hospital on the tenth day following the operation.

News was had of him ten months later. He still had the seizures about once a fortnight, and was still hemiplegic with paralysis of the left leg, but the movements of the left arm were normal, although awkward. He also had a loss of memory and had frequent headache and vertigo.

Finally, in some cases the bullet may be deformed from ricochet, in which case it acts like a piece of exploding shell, and this brings us to the consideration of wounds of the head from artillery projectiles. These are of two kinds: the fusing shells and the percussion shells. The former explode at a certain height and are charged with small lead bullets; these are shrapnel. They produce a rounded aperture, and this is the only thing that distinguishes them from wounds produced by exploding shells.

Percussive shells explode in contact with the ground. They are made of a steel envelope of some thickness, containing an explosive charge. When they burst, the metal envelope becomes fragmented into quite a number of pieces, whose shape and size vary. Some are very large, weighing as much as several pounds, in which case they produce such extensive lesions to the skull that death is instantaneous. The lesions that they cause are different from those resulting from bullets, on account of their lesser velocity and irregular shape.

When they come in contact with the scalp these bits of shell usually produce irregular wounds with jagged edges. In contact with the skull they produce variable lesions, according to their size and velocity, from simple contusion to complete perforation of the skull, but it is most uncommon for them to go through the head.

Contusion is common. On the external aspect of the skull an area

of hemorrhagic spots or points will be seen which give issue to blood as fast as they are wiped. Sometimes there is a real ecchymosis.

But usually, under this minute lesion of the external table, there are more marked injuries to the internal table, the latter being most often fractured. The fragments project toward the brain and may directly damage the cortex.

Underneath the contused bone fissures will often be found if looked for. They may be limited to the area involved, and this is relatively frequent in injury from exploding shells, but I would particularly insist on the fact that they *always involve both tables*. For example, a soldier presented a localized crack in the right parietal bone, involving the entire thickness. However, such fissures may extend quite a distance, even to the base of the skull. In one case the patient presented a fracture of the right parietal and temporal bones, with fissures extending to the base.

In the following case there was a fissure, resulting from a wound situated in the apex of the skull, which extended transversely along the entire vault, involving both parietals and temporals, and on the left extended to the optic foramen.

When the patient was brought to the ambulance he was found to have a number of scalp wounds, which did not seem to be penetrating. He did not show any functional sign, excepting that he was mentally dull. The injury resulted from an exploding shell.

On the following day the general condition was much the same, but a left-sided palpebral and conjunctival ecchymosis was noted, likewise unequal pupils. In the evening the temperature was 102.1° F. and the patient was delirious. He died the next morning in coma with a temperature of 105.4° F.

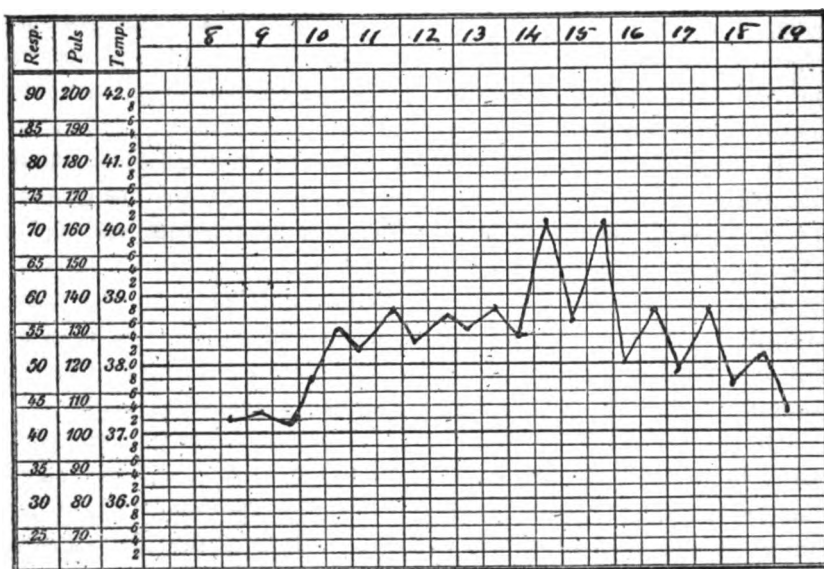
At autopsy there was an abundant blood collection under the epicranium. One of the wounds, located almost exactly in the median line of the left parietal, involved the bone. It was linear and about one centimetre long, and involved both tables and the dura. From this wound a long transversal fissure started and extended over to the right as far as the temporal region. On the left it extended to the base of the skull, up to the optic foramen. After removal of the vault an abundant collection of blood was found spread over the brain, which itself appeared intact.

The third degree of bone lesions is represented by depression, the

missile acting exactly like the blow from a hammer. The internal table first gives way, and then the external table, as, being no longer supported, it becomes depressed. The depression may be formed by a single fragment, as in the following case, which presented a depression of the external table of the right parietal bone about the size of a walnut.

When the patient arrived at the ambulance a small, irregular wound was found in the right parietotemporal region and a tunnel of the neck in the sternomastoid region, due to exploding shell. Two

CHART 1



days later, under chloroform narcosis, an incision was made in the scalp and a good-sized piece of shell removed. A small depression in the external table was then seen, about the size of a ten-cent piece; the internal table was fractured also. The opening in the skull was enlarged and four splinters of bone were removed from the internal table. The dura was intact and pulsated normally. A wick was inserted and the incision sutured.

The temperature, which was 99.4° F. in the morning, was 101.1° F. in the evening.

The following day (September 11) the general condition was

excellent, and there is the temperature chart of the case (see Chart I), and if you will follow the curve and dates, I will tell you what happened. Let me first say that the head wound was dressed daily and remained perfectly normal, and there was at no time the slightest trace of suppuration, but on September 15 there was some erythema on the thigh, in the region where the antitetanic serum had been injected, while there was a generalized scarlatiniform eruption over the body. The patient had simply developed a scarlatina, so that on September 19 he was transferred to a hospital for contagious diseases in the rear.

On February 26 following, a letter was received from the patient in which he stated that he remained at the contagious hospital with a high temperature and was given his discharge on December 28. He said he was perfectly well, and, although for a time he had had intermittent headache, this had completely disappeared.

Often there is only a simple bone splinter from the internal table, but more commonly the depression is composed of a number of splinters of variable size and shape, but generally they are triangular. These fragments are more or less adherent around the depression. They give rise to a depression in the external table and form a pyramid projecting toward and cutting into the dura. Such a condition is frequently met with.

For example, in one case a small depression in the right parietal was found, with multiple fragments on the internal table; another case showed at operation a little depression in the left parietal and a fracture with large splinters of the internal table. The patient was brought to the ambulance on September 26 with a wound of the left frontoparietal and numerous wounds on the back and left arm, the result of an exploding shell. Upon arrival a paralysis of the right arm was noted.

Operation was done at once under chloroform narcosis. After a crucial incision of the scalp a depression in the external table was discovered over the left frontoparietal region. The opening was enlarged with bone-cutting forceps, and the splinters from the internal table were removed. The dura was intact. Gauze wick. Flat dressing.

After the patient was returned to bed he was given salt solution by Murphy's method. On the following day the wick was removed,

and on the next day the patient was transferred to another ambulance. Five months later the patient was still in a base hospital. He was well, but was afflicted with paralysis of the right arm.

Depressed fractures of the skull are frequently accompanied by fissures, running in various directions, sometimes extending to the base.

Finally, an exploding shell may produce a complete perforation of both tables, in which case the fragments of bone are driven more or less deeply into the brain or carried in by the projectile. The resulting wound is very irregular and of variable size, and the borders of the perforation are never clean-cut, as in the case of a bullet wound. For example, a patient presented a large, straight wound with anfractuous borders in the left temporoparietal region, while in yet another the loss of bone over the right temporo-occipital region measured about 12 centimetres in length. On the contrary, in still another patient the aperture was very small. I will briefly relate the case-histories of these three patients.

The first patient entered the ambulance on October 14. There was a scalp wound from an exploding shell which also involved the cranial vault in the left temporoparietal region. There was a slight paresis on the right side.

On the following day, under chloroform narcosis, a crucial incision of the scalp was made over the wound, which exposed a broad, straight opening in the skull with irregular borders. This opening was evened off and the bone splinters removed. Blood-clot mixed with mashed brain matter came out of the opening. Wound cleansed; wick drain, aseptic dressing.

After the operation the paresis was found to have disappeared. The wound, which was not infected at the start, suppurated a little, but the temperature remained normal.

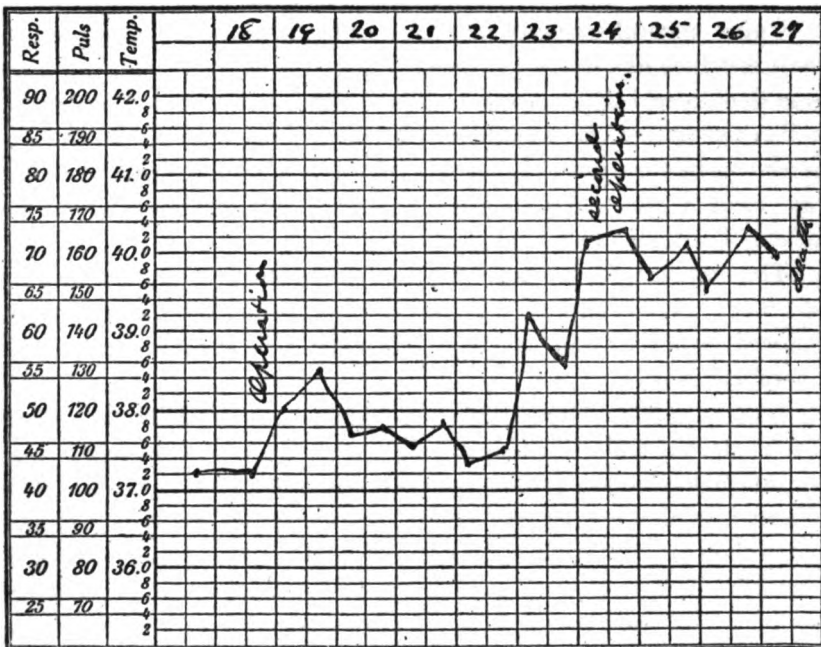
On October 18 a radiograph was taken, which showed that there was no intracranial projectile. On October 29 the patient was transferred to another ambulance, where, on November 10, a second operation was done for cerebral abscess and cerebral hernia. After this interference the patient presented a succession of buttonhole cerebral abscesses, complicated by hernia of the brain. Several of the abscesses were incised and a portion of the cerebral hernia was resected.

On January 6 the patient was transferred to a base hospital in good condition, but he died the following month.

The second case was that of a soldier who entered October 18 for a penetrating wound in the right temporo-occipital region, with issue of brain substance. There was a left hemiplegia.

Under chloroform anæsthesia the scalp was incised and a wound in the skull exposed. It was about 12 centimetres long and two centimetres wide, and blood-clot and mashed brain substance made their

CHART II



exit through it. The splinters from the internal table were removed and the edges of the bone aperture were evened off. The wound was cleansed, a wick drain inserted and a flat dressing applied.

I here offer the temperature chart of this case (see Chart II) and will tell you how the affair ended.

On October 19 the hemiplegia was less, but the patient said he could not see. On the 20th he could see with the right eye. On the 23d there were some epileptiform seizures—eight in the forenoon and two in the evening. Some delirium.

On October 24, under general narcosis, the wound was again cleansed and a cerebral puncture was made with a long needle, but no pus focus could be discovered. On the following two days the seizures were more frequent, and on October 28 the patient died in coma.

The third case in question was that of a soldier who entered December 15 for a penetrating wound in the left temporal region from an exploding shell. The patient was not wearing his helmet when hit. There was a small wound with a large hæmatoma and issue of brain substance. The patient was semi-comatose.

Under general narcosis a crucial scalp incision was made and a small aperture in the skull exposed. This was enlarged and splinters from the internal table removed. There was a perforation in the dura, so this membrane was incised and a small divided artery in the pia mater was ligated, as it was bleeding freely. A wick soaked in alcohol was inserted and a flat dressing applied. After the operation the patient was found to be aphasic.

The outcome of the case was that the aphasia improved, and on December 28 the patient was transferred to an ambulance farther removed from the front, where, on January 6, he was operated on for a cerebral hernia.

Lesions of the internal table are always more extensive than those of the external table. The bone splinters which become detached from the internal table are frequently very large, but remain more or less adherent to the rest of the bone. For example, in one instance, under the opening in the external table, which was about two centimetres in diameter, many splinters were found on the internal table, two of which were as large as a silver quarter dollar. I will give you a brief account of the case.

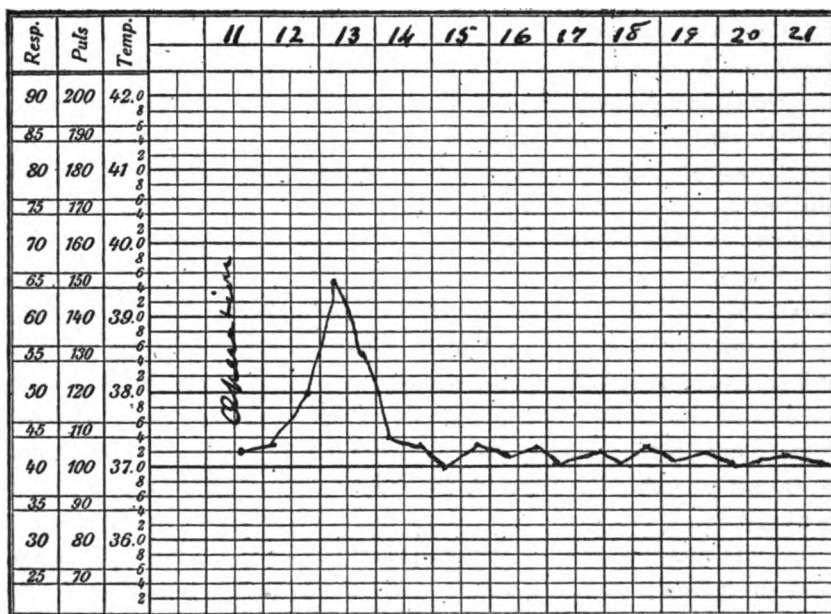
The patient presented a large transversal scalp wound in the occipital region. Other than some mental dulness, there were no functional symptoms. The wound was immediately enlarged, and upon exposing the skull an opening about two centimetres in diameter was discovered. This was enlarged with bone forceps and the above-mentioned splinters projecting from the internal table into the brain were removed. The dura was freely incised and a loss of cerebral substance about seven centimetres deep was discovered. This was irrigated with artificial serum. A gauze wick was inserted and a

flat dressing applied. The next day the wound bled freely and the dressings were changed.

For the first two days following the operation the patient was comatose and the temperature was up for several days, but finally went down. This is the chart (see Chart III). As the ambulance left the village where it was stationed on March 21 the patient was transferred to another ambulance, and on April 26 a letter was received from the patient, stating that he was in very good condition.

Attention should be directed to small bits of shell which produce

CHART III



lesions of a special type and which Leriche has very ably described. A small dark depression in the bone is found, never larger than a cherry-stone, usually much smaller, with deep splinters. These small bits of shell may even traverse the bone, as in the following case, in which there was a very small wound in the parieto-occipital region and which was found to be a penetrating wound, the dura having been perforated.

The patient was brought to the ambulance on November 23 for multiple wounds on the back and scalp from bursting shells. The

wounds were explored under general narcosis with ethyl chloride and a very small penetrating wound was found in the left parieto-occipital region. The bone presented a minute blackish orifice.

The narcosis was continued with chloroform and the crucial scalp incision was enlarged. The tiny opening in the skull was enlarged with a Doyen drill and with cutting forceps. The splinters from the internal table were removed, and then a very small slit in the dura was discovered, which was enlarged. The underlying cerebral surface was cleansed and a gauze wick inserted.

On the following days the general condition remained good, the temperature fluctuating around 100.2° F.

On December 3 a radiograph was taken, but did not reveal any intracranial projectile.

December 8, morning temperature 102° F., evening 102.4° F. Some cough.

December 9, temperature still up, wound in good condition. Some dulness at left pulmonary base. Pleural puncture withdrew a small amount of serofibrinous fluid.

For the next five days the temperature remained up, and on December 14 it was still at 100.2° F., although the pleuropulmonary symptoms had cleared up. Under general narcosis the scalp wound, which had cicatrized, was opened and the trephine opening enlarged. A crucial incision was made in the dura and an area of sclerosis was found in the brain. This was incised and a cavity the size of a walnut exposed, but which contained no pus. Cavity lightly packed with gauze.

After this interference the temperature fell and remained normal, and the patient was transferred to a base hospital in good condition on December 28.

The lesions of the base of the skull are rare when compared with those of the cranial vault. The projectile penetrates by one of the anatomical cavities—the orbit, mouth, ear—and produces severe lesions in most cases which result in death.

You must also remember that the cranial bones are very vascular and therefore bleed freely. A blood infiltration is always found in the diploë which may reach such proportions as to form a true hæmatoma. But the hemorrhage may take place into the supradural space, as in the following case.

The patient was brought to the ambulance on November 25 for wounds of the thumb and forearm and a scalp wound over the left parietal region, the result of an exploding shell. There was no functional symptom of penetration. Temperature, 99.4° F. The patient was wearing his helmet at the time of the injury.

Under general narcosis a scalp incision revealed a depression the size of a fifty-cent piece in the right parietal bone. The splinters coming from the internal table were removed, some being quite large. The dura seemed to be intact, but the supradural space was filled with clot over quite a large area. Gauze drain.

The patient recovered and had a perfectly normal convalescence. On December 3 a radiograph was taken with negative results. The patient was sent to a base hospital on December 21.

I shall now refer briefly to lesions of the brain and dura. The latter, from its anatomical situation, is exposed more or less to injury, according to the location of the cranial lesion and the violence of the trauma. You will remember that the dura is not closely adherent to the inner table of the skull at all points, and, although at some spots it is so, at others it is much more lax. The dura adheres very little in the so-called detachable area which extends from in front backward to the posterior edges of the lesser wing of the sphenoid, three centimetres from the internal occipital protuberance, and from above downward, from the superior longitudinal sinus to a line joining the lesser wings of the sphenoid with the base petrous portion of the temporal bone, representing an area measuring about 12 centimetres. The dura adheres quite intimately from the side of this area to the remainder of the vault, particularly in the region of the sutures, but its strongest adhesion is to be found over the prominences of the base and the points of exit of the cranial nerves.

When a projectile has traversed the bone, it acts in one of two ways. At those areas where the dura is adherent it will be perforated at the same time as the internal table, while at those areas where it is loosely held over the inner surface of the inner table the projectile may simply press it back without perforating the membrane. When this happens the projectile may be found lying on the dura, usually in the midst of a hæmatoma.

The bone splinters from the inner table will act in the same way, and therefore it is clear that the detached splinters from the inner

table will perforate the membrane in the areas where it adheres and only push it downward at the other areas. When the dura is perforated the opening will vary in size and shape from one case to another, according to the nature of the projectile. When the missile goes through it the resulting aperture has about the same shape and size as the projectile, but it may be very small when the perforation is due to a tear from a splinter-point. When the dura is pushed downward by a projectile or splinter it is quite apt to present an area of superficial contusion, in which case it takes on a red, ecchymotic hue instead of offering its normal bluish color.

The brain itself is more prone to be injured in through-and-through bullet wounds. Here the entrance wound is conical in shape, with its base external, this being due to the scattering of the splinters, but throughout the remainder of the tract the lesions are extensive, the surrounding structures being much contused over quite a considerable extent. When the shot is at short range the skull bursts open and the brain gushes out in the form of a pulp. When bits of exploding shell penetrate the brain the resulting lesions are the same as in bullet wounds, but the disorders produced are often more marked on account of the size and irregular shape of the missile.

When the projectile does not perforate, as in the case of a bullet striking tangentially or a bit of shell located outside of the dura, the brain presents a contusion usually larger in extent than that of the wound in the skull. *These lesions may also exist without there being any injury to the dura.*

Cerebral contusion, when slight, shows itself in the form of an interstitial hemorrhagic network, but, when more severe, more extensive blood suffusions are encountered, forming dark red or brownish areas, surrounded by a reddish border. Blood-clot will be found in the sulci, while a suffusion of blood covers over the convolutions. Blood may be found in the ventricles.

Such hemorrhagic lesions are accompanied by destructive lesions, which arise on account of the tenuity of the brain substance, whose fibres and cell strata are disorganized by the shock and blood extravasation. You then will find a sort of reddish-brown bloody pulp contained in the irregular foci, whose walls are riddled with miliary hemorrhages.

The other structures of the cranial contents may also be injured,

particularly the vessels and nerves. The sinuses are frequently involved, either by being torn open or perforated by bone splinters. The superior longitudinal, lateral, and cavernous sinuses are those commonly involved.

The resulting hemorrhage usually stops of itself, as the aperture becomes occluded by clot or is closed by the projectile or bone splinter producing the wound, as is illustrated by the following case.

The patient was brought to ambulance for a large penetrating wound in the frontal region in the middle line, with issue of brain substance. He was dazed by the exploding shell, remained lying down for half an hour at his post, and then, as his wound bled freely, he went on foot to his regimental dressing station. There the wound was dressed and a bone spiculum about two centimetres long and one centimetre thick was removed. After this the patient went on foot for about four miles in the trenches to the rear dressing station, and from there was sent to the ambulance in an automobile.

The wound in the frontal region was rounded and about four centimetres in diameter, at the bottom of which some spicula were seen, as well as brain substance mixed with clot. He presented no functional symptom, but appeared stupid.

General chloroform narcosis. The wound was enlarged by a crucial incision of the scalp, and a large opening in the skull was exposed, through which brain matter extended. The numerous bone splinters were removed, and then a severe hemorrhage arose from the superior longitudinal sinus. The wound was cleansed and packed with gauze. Shock treatment was given after his return to bed, but coma developed and the patient died the following morning.

The middle meningeal artery may be wounded and give rise to hemorrhage, which may be external, or produce a hæmatoma, either over or under the dura, according to whether or not this membrane has been perforated. The same applies to the vessels of the pia mater.

Lesions of the cranial nerves may be met with, but are of little interest surgically; but what you must not forget, because of its great importance from the standpoint of infection, is that the projectile always carries bits of clothing, hair, etc., into the tract along with it, and their removal is imperative; otherwise, severe infection will occur and carry off the patient.

Certain injuries of the head are instantaneously fatal on account

of the formidable extent of the lesions or from some rapidly developing complication, such as commotion, contusion, compression, or severe hemorrhage.

In contrast to these cases, other patients recover without the slightest untoward accident and with great rapidity. Then you have those cases where the *initial evolution* appears excellent. The wound closes, the patient appears to be recovering perfectly, and then, after several weeks or months, sometimes a year or more after receipt of the injury, infectious accidents develop which kill the patient more or less rapidly. The cause of this is usually the inclusion of the projectile somewhere in the wound or secondary migration of the missile.

I would also mention certain mental or nervous disturbances, such as epilepsy, paralyses of various types, and headache, which may arise a long time after apparent recovery from a head injury. The conclusion, therefore, to be drawn is that all foreign bodies must be removed at once, and the projectile likewise, when it is not eliminated spontaneously.

What, however, is yet more important, from the standpoint of immediate surgical treatment, is the evolution of the cranial lesions, which I shall now describe in some detail.

The first few days following the receipt of the injury usually offer nothing of any note. The patient is apyretic, recovers from the initial coma, and enjoys a perfect euphoria. He arises, eats, walks, and does not suffer. The wound progresses favorably. Then, without any apparent prodromata, at the end of the first week, sometimes at the end of a fortnight or even much longer, an acute meningitis causes sudden death.

Such is the ordinary outcome of some cranial injuries which after a period of quiet simulating a favorable evolution suddenly develop a meningeal syndrome of rapid and fatal outcome.

Now, to be forewarned is to be forearmed, and since you know that a meningo-encephalitis may develop you must always be on your guard during the operative convalescence of these cases. To prevent its development is the end to be attained by immediate operation in every case of cranial injury, no matter of how little consequence it may seem when the patient is just examined, and after this your object must be to detect the very early manifestations of a meningitis

in order to resort to proper therapeutic measures in seasonable time. Your line of action must be derived from the study of its causes, evolution, and symptomatology.

Let us first consider the frequency of *meningitis*. Without any exaggeration whatsoever, you may take it that *it is constant in every case where the dura has been perforated*. In some instances of tear of the dura situated under a fissure in the skull, an immediate surgical interference will allow you to cleanse the superficial focus properly, so that the traumatic aperture in the dura can be considered aseptic and dealt with as such, but I hasten to add that such cases are very uncommon.

Just so soon as a lesion of the dura is exposed or of some little standing, the injury will surely be complicated by infection. All degrees of infection exist, from serious meningo-encephalitis, with rapid generalization and death, to a mild local reaction, followed by spontaneous recovery, merely offering vague symptoms which must be sought for in order to be detected. These varying degrees of the process are clearly the consequence of the extent of the mechanical disorders and the factors of infection.

There is one condition which would seem to be essential for the development of meningitis: I refer to a solution of continuity in the dura. Through an aperture, however trifling, caused by a spiculum of bone or the projectile itself, the agents of infection will find their way, and the danger will be all the greater, the more gaping, contused, and irregular the wound.

The question has arisen whether or not a suppurative process can arise *under* the dura without any solution of continuity in this membrane, and some cases appear to show that such conditions may be met with. To explain this, two hypotheses have been put forward, the first and rather improbable one being by way of the circulation, starting from a focus of cerebral attrition or a subdural hæmatoma.

To my way of reasoning, however, it is more probable that there always exists some minute lesion of the dura, so small that it is likely to be passed over at the operation. Such lesions are erosions, fissures, or an invisible prick in the membrane, resulting in an immediate infection, all the more to be feared because the superficial structures are the most infected and our therapeutic measures make it a material

impossibility to aseptinize the depths of the wound through so small an aperture.

Whether limited to the meninges or also involving the brain, the infectious process is, at the beginning, always localized. Later it becomes generalized, but, whether local or general, the infection arising in the arachnoid spaces produces the well-known forms of meningitis; namely, the serous and the purulent. The latter form is by all odds the more frequent.

The *pathology* of these meningitides deserves to be thoroughly studied. Among other things it would show the frequency of, and the part played by, adhesions in the extension of the inflammation, but as yet we are ignorant as to their points of election and the factors favoring their production.

Suppurative or otherwise, the meningitic process is, in the beginning, always located around the entrance wound. It may remain confined to this area and be recovered from after the evacuation of the focus, when the case is the suppurative type.

The adhesions covering an area of cortical hyperæmia, which is the cause of many anxieties, are generally the consequence of the recovery, and are the residue of the acute pathologic process.

The septic process may extend and become progressively generalized, a condition frequently met with. The factors favoring the extension of the process appear to be many, the first being a retention of the septic fluids. It would seem reasonable to suppose that when the subarachnoid spaces have been opened the resulting spontaneous flowing off of the cerebrospinal fluid ought to produce a real cleansing effect by bringing away the septic matter and thus acting as an irrigation from within outward of the contaminated area.

But, gentlemen, this discharge of cerebrospinal fluid does not take place, and consequently many factors, such as hemorrhage, congestion, cedema and intracranial hypertension, which immediately arise after the injury, force the brain against the entrance wound in the skull, thus occluding the aperture opening into the subarachnoid space.

The beginning of a cerebral hernia soon manifests its presence, the spontaneous escape of the infected cerebrospinal fluid and the succeeding suppurating collection become impossible, and the result is clearly a retention, so that the localized area of meningitis will not

be slow in extending, and this leads me to the question of the factors which favor this extension.

It seems rational to suppose that the patient's position must unquestionably exercise some influence on the extension of the meningitis, just as it does in the case of a septic peritonitis—of course, taking into consideration the essential anatomical and physiological differences between the two serosa. I can not see why it would be illogical to assume that the direction and rapidity of the progression would be different when a patient is lying down with the head inclined on the side of the injury and a patient with the thorax elevated and the head resting on the sound side.

The progression of the inflammatory process taking place step by step in the subarachnoid network would seem to indicate that the cerebrospinal fluid is an important factor of this progression. With it, the infection will preferably follow the principal routes that anatomy of the cerebrospinal system teaches and which converge toward the large venous dilatations of the base. The generalization is then accomplished, extension to the ventricles and spinal canal is inevitable, while the prognosis is no longer doubtful.

Extension of the septic process to the base is evidently the natural tendency and is the real danger of a localized meningitis due to a projectile. Everything being equal, the immediate prognosis should clearly be all the better the higher the injury is seated in the vault and therefore more remote from the base.

Encephalitis accompanies the meningitis. The suppuration of the focus of cerebral attrition resulting from the passage of the projectile is more commonly termed cerebral abscess, a fact which gives rise to some confusion. The characteristics of this particular intracerebral suppuration are so peculiar to themselves that I think the process would be more aptly called "suppuration of the cerebral traumatic focus," retaining the term "cerebral abscess" for those collections which apparently are not in direct relation to the action of the missile.

A focus of cerebral attrition will undergo a different evolution according to its location and extent, its connections, the nature of its contents, and the degree of infection. When it is superficial under an intact dura, its evolution may be aseptic and give rise to only vague symptoms of mild degree if the area involved is small. On the other

hand, it may suddenly develop suppuration at a rather late day, and you must always bear this eventuality in mind.

When a projectile penetrates the brain infection is sure to arise, as the missile must be considered septic in every case and carries other foreign bodies along with it whose septic properties are generally great, particularly hair, bone spicula, bits of clothing, etc., with the result that the projectile floats, so to speak, in a diffuent mass composed of mashed cerebral substance and more or less coagulated blood.

The walls of the cavity, which has no distinct boundaries, are composed of nerve-tissue which has undergone certain changes, usually being softened, several millimetres in depth, and then by a slow gradation apparently normal structures are reached.

The softened area is bound to undergo necrosis, and its liquefaction will ultimately increase the size of the cavity in some instances to such an extent that communication with a ventricle or an opening into a subarachnoid confluent may result. The infection adds its effect to the increase in size of the cavity by liquefaction of its walls, while continued tension acts in the same way.

An infected, suppurating focus of cerebral attrition should empty itself—naturally in the direction of lesser structural resistance. Therefore it will be in the direction of the exterior, if the apertures in the dura and skull are sufficiently large not to cause strangulation of the resulting cerebral hernia; otherwise the focus will extend to the subarachnoid space or a ventricular cavity.

The opening into a ventricle will be all the more likely the larger the two cavities are, as they are merely separated by a thin layer of necrosed cerebral tissue. There are also cases where a communication with a ventricle occurs at once as the result of the projectile's course, and the gravity of this condition is evident. In opposition to these very serious cases, you will meet with others in which the virulence is greatly lessened and where the contents of the focus attrition are tolerated for weeks or even months, and give evidence of their existence only at a very late date.

Instances of this kind are offered by cases of suppuration around a projectile embedded in the brain, where the tract and aperture of entrance in the skull and dura have undergone a process of cicatrization. In both instances suppuration of the focus of attrition assumes

the clinical aspects of a cerebral abscess, quite independently of any immediate external cause.

What essentially differentiates suppuration of a focus of cerebral attrition is its communication with the exterior by the tract produced by the projectile which will permit of its spontaneous evacuation and consequently its repair and cure if dealt with surgically. Collections which are independent of the bullet tract must be very uncommon, and I even doubt that they can occur.

I would sum up this subject thus: Infection of a penetrating wound in the brain occupies two distinct areas, namely, the sub-arachnoid spaces, which means meningitis; and, secondly, infection of the cerebral substance, which means suppuration of the focus of attrition.

There is not much to be said in respect to the *symptomatology* of meningo-encephalitis following gunshot wounds of the head. The process usually develops clinically in from one to three weeks following the receipt of the injury. Besides the purely depressive type with headache, somnolence, and coma, you will sometimes meet with the classic type with headache, vertigo, nausea, high temperature, Kernig's sign, stiffness of neck, changes in the pulse preceding the advent of paralyzes in the cranial and spinal domains, and, finally, coma.

It is in this latter type that you will meet with certain forms of convulsion, contractures, and paralyzes which indicate the localization of the meningeal exudates over such and such areas of the cerebral convolutions. Ophthalmoscopic examination for optic neuritis, etc., will often give valuable diagnostic data.

This likewise applies to the blood examination, which will reveal an increase of the white cells, particularly the polynuclears (95 per cent.), while analysis of the cerebrospinal fluid shows pus or polynucleosis; in other words, leucocytic evidences of suppuration.

Let me also mention, in respect to the evolution of this usually fatal process, that there are cases where death supervenes without any previous symptom occurring, and also those which are absolutely overwhelming in evolution, probably the result of a purulent ventricular inundation. What I desire to particularly impress upon you is that it is quite unnecessary to await the complete symptom-complex for making the diagnosis of meningitis. On the contrary, it is incumbent upon you to make it at the very beginning, at a time when

surgical measures may be still of some avail, and in order to attain this end you must systematically look for the elements of the *meningeal syndrome*; namely, Kernig's sign, stiffness of the neck, headache, and bradycardia.

It is only by daily observation of these cases, thus minutely following the evolution of the process, that you will be able to estimate the meningeal reaction, and therefore the prognosis. If you follow this advice you will be unlikely to overlook a commencing meningitis in the midst of improving cerebral symptoms, such as coma or paralyses, an amelioration due to the happy effects of an early and properly conducted surgical interference—a most important fact, because the future of a subject having sustained a cranial injury, when the initial trauma is compatible with life, above all depends upon the element of infection, which clinically is made evident by the meningeal syndrome. Therefore the logic of all this is to detect these elements at their first appearance and follow their progress, which is your surest guide for your conduct of the case. But, regardless of our therapeutic resources, it must be admitted that meningo-encephalitis is a very serious complication, and also that it is the causal factor of still another accident in the evolution of head injuries. I refer to cerebral hernia, which I shall now consider.

Hernia of the brain is a relatively frequent complication of meningo-encephalitis, and is the result of the infection of the cerebral structures. Its causes are congestion and oedema of the brain, a portion of the organ becoming mechanically forced out through the cranial opening whenever intracerebral hypertension exists, and you know that hypertension always accompanies infection. There is a decided increase in the production of cerebrospinal fluid and formation of a meningeal or cerebral abscess.

This phenomenon occurs during the first fortnight following the receipt of the injury. The projecting cerebral mass is soft and may or may not present pulsation, and will increase in size when the patient coughs or makes some exertion. It is more commonly composed of cerebral substance, and may contain a serous fluid or pus. It varies in size from a walnut to a closed fist, while the functional symptoms to which it gives rise are those of localization and are too uncertain to be of much, if any, value.

The evolution of hernia of the brain is interesting. The mass

may become eliminated by a process of necrosis, and this occasionally gives rise to hemorrhage when pieces of the hernia are shed. The hernia may slowly and progressively become reintegrated with a complete epidermization, or, on the other hand, reintegration may take place suddenly after evacuation of a neighboring collection or by the removal of a certain amount of cerebrospinal fluid. Finally, it may persist.

Hernia of the brain is usually nefarious, as it forms a plug and occludes, by the pressure it produces, the opening into the sub-arachnoid space, thus preventing drainage. If it involves the area traversed by the projectile, it occludes the track which otherwise would assure the normal evacuation of the focus of cerebral attrition, and consequently provokes mechanical retention.

Therefore it becomes evident that the prognosis is serious, both because it governs the intracranial hypertension, which itself is due to infection, and because it mechanically prevents outward drainage of the products of this infection.

Free discharge of cerebrospinal fluid and severe hemorrhage are yet two other complications to which consideration must be paid, as they are relatively frequent in cranial wounds from projectiles of war.

The first of these, which takes place by way of the tract produced by the missile, is either in such small amount or so completely mixed with bloody discharge that it may very likely escape notice. Various factors govern or prevent this discharge from occurring. Such are the narrowness of the wound, its high situation, in the neighborhood of the cranial vault, or the obstruction offered it by a hernia of the brain or a plug of mashed cerebral substance. If the head is raised, this also prevents its escape. Naturally a large aperture situated low, a declivous position of the head, coughing, etc., are prone to induce the outward escape of the cerebrospinal fluid, and when it is in sufficient amount it should be collected for examination. This is interesting, from the viewpoint of diagnosis of deep-seated initial lesions, when blood will be detected in it or for future complications when a polynucleosis is present, as this is a sign of infection.

A free discharge of cerebrospinal fluid does not seem to be dangerous, so far as the ultimate condition of the patient is concerned. It sometimes even reduces the temperature permanently and acts as

an irrigation of the track of the projectile from within outward, and likewise carries away the toxic matter from the meninges or brain.

If, for any reason, its discharge should become alarming on account of its amount, the head should be elevated, and this will usually control the situation.

As to hemorrhage, it may be either primary or secondary. The former depends upon the wound itself or arises during trepanation. Injury to the meningeal or cerebral arteries, the sinuses, or internal carotid are frequently fatal, and, as I have already pointed out, the projectile or bone spiculum may produce hæmostasis for the time being by plugging the opening in the vessel.

Secondary hemorrhage is the frequent result of infection or during the necrobiotic process in a brain hernia. It may be sufficiently profuse to be serious, but this is rather more likely to be due to the recurring frequency than to the actual amount of blood lost each time. The prognosis is serious, if only by reason of the cause of the hemorrhage itself, which keeps up the loss of blood.

It hardly seems necessary to speak of the prognosis and diagnosis of injuries of the skull and brain from projectiles of war, because, from what has been said, I think you will be able to deduce your conclusions in this respect without further comment on my part. Therefore my next lecture will be devoted entirely to the treatment of these lesions and their complications.

(To be continued)

CLINIC ON MULTIPLE NEURITIS

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GENTLEMEN: Multiple neuritis is an organic disease of the periphery of the nervous system, but in importance it ranks at the head, for it is quite common in our busy centres. And not only because of its frequency, but also because of its favorable prognosis, does this disease deserve careful attention. We are therefore fortunate in the fact that the Cook County Hospital has such an abundance of material at our disposal that I am compelled to exercise some self-restraint not to yield to the temptation of showing too many cases. My plan is to present a fairly typical case of each of the more common varieties of the disease, and at the close of the entire presentation to discuss the disease in its general features, having due regard to diagnosis and treatment.

CASE I.—The first patient is a Russian laborer, whose English is too deficient to give a complete history at one sitting. From the several subsequent conversations we extract the statement that three weeks before his admission into the Cook County Hospital he began to suffer from distressing pains in the legs and hands, difficulty in walking, and peculiar sensory manifestations to be described later. Up to the beginning of the present trouble he claims to have been in good health and free from venereal infection. He boasts of exemplary habits, though admitting the occasional use of tobacco and whiskey. Later he confessed to indulging regularly in small quantities of strong drink, though not to the point of becoming intoxicated.

Examination.—Observing the patient's gait as he walks across the amphitheatre, you will notice that it is peculiar, in that the steps are not regular and there is a tendency for the feet to remain on the floor. In order to clear the toes from the ground—to overcome the toe-drop—he is compelled to raise his knees high, thus imitating the high-stepper's gait, which gave it the name of "steppage gait." The

muscular weakness is insignificant in the thighs, but becomes more marked from above downward, so that the foot is almost without power and drops of its own weight unless supported. The important point is in the phenomenon of greatest involvement of the peripheral extensors. A condition similar to that of the lower extremities prevails in the muscles of the upper extremities. The hands and arms are extremely weak and patient is unable to force the lever of a dynamometer, an instrument used for the measuring of muscular power, from its zero mark. Both hands are habitually flexed at the wrist because the extensors are paralyzed and permit the hands to drop into the palms—a good example of so-called wrist-drop. Not only is extension impossible, but even flexion is very much reduced in the hands, because it lacks the coöperation of the extensors. That defective flexion is the result of lack of extension I can demonstrate by placing my hand on the back of the patient's wrist, thus replacing his own extensors—and immediately the flexion becomes normal. The toe-drop and wrist-drop are well shown even in the accompanying photograph (Fig. 1) taken of the patient after improvement had already begun.

Subjectively, there are occasional spontaneous pains, especially in the legs and arms, described as sharp and darting, but he complains of another kind of sensory discomfort, not amounting to pain, more or less constantly present—an aching which annoys him more than the occasional sharp pain. Objectively, the slightest touch applied to the skin of upper and lower extremities, even mere contact of the bed-clothes with the skin, evokes unpleasant sensations. The climax of his suffering is reached when we apply pressure over the calf-muscles, the Achilles tendons, or the arm muscles. This symptom of pain in muscles on slight pressure is a valuable differential sign for this disease, as it is found in but few other conditions.

A rather interesting sensory symptom present in my earlier examinations of the case was the so-called "delayed conduction of sensation"; that is, when the skin is pricked with the point of a pin the patient does not respond immediately, but the answer is delayed because of the delayed conduction of the impulse along the nerve. This symptom is not uncommon, and for long has been considered pathognomonic of tabes; we now know that it may be present in multiple neuritis before the development of anæsthesia.

FIG. 1



Showing the characteristic foot-drop and wrist-drop of multiple neuritis, as well as the wasting of the peripheral parts.

Palpating the nerves where they have become superficial, as for instance the ulnar nerve between the olecranon process and the internal condyle, gives a ready response of pain. This is a finding directly opposite to that in tabes, in which disease there is mostly complete absence of pain on pressure of the ulnar nerve-trunk, a phenomenon first described by Biernacki as characteristic for that disease and since known as *Biernacki's sign*.

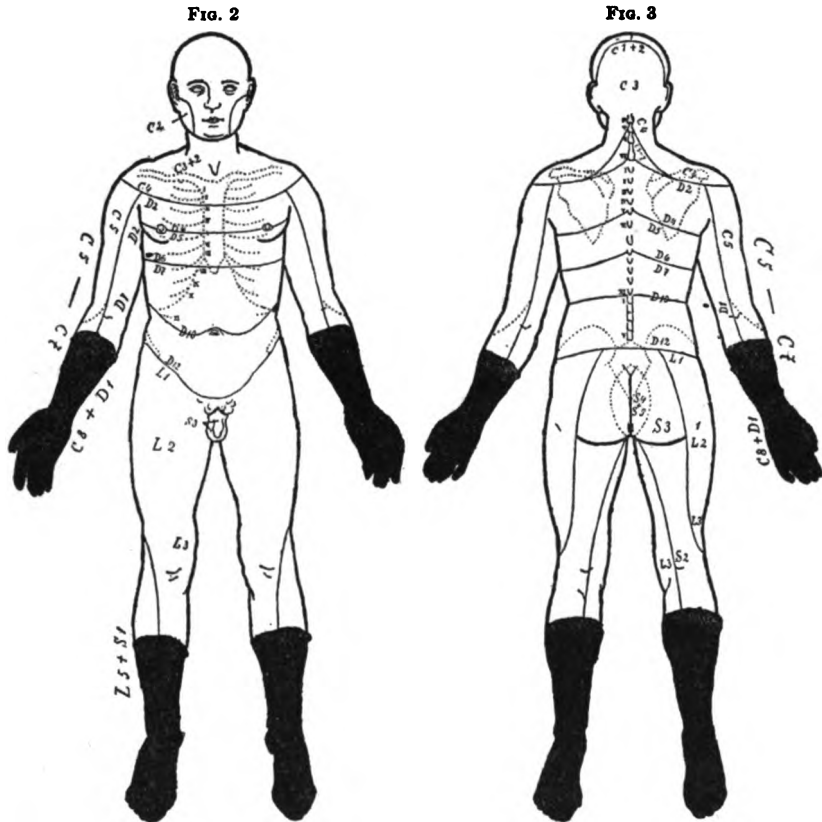
Continuing the examination of the sensory portion of the nervous system by the ordinary method of employing a camel's-hair brush and the point of a pin for the determination of the sense of pain and touch, respectively, we find the hands and forearms irresponsive to both pin-prick and touch over the parts shaded dark in Figs. 2 and 3. The anæsthetic area so depicted is almost glove-shaped in the upper extremity and very much resembles a stocking in the lower extremity. It is well to note that the glove and stocking anæsthesias, bilaterally and symmetrically distributed, constitute a well-known syndrome of multiple neuritis.

The Reflexes.—The superficial reflexes, such as the abdominal and plantar, are somewhat reduced, but not pathologically; the characteristic change, however, is in the deep or tendon reflexes. Of these the triceps gives a bare response, the wrist-tap none at all; the knee-jerks are bilaterally absent; likewise the Achilles reflexes, which were faintly present at the first examination. In short, all the deep reflexes connected with the dependent or peripheral parts of the body seem to have practically disappeared.

Completing the examination, we find pupils which respond to light and accommodation, though somewhat sluggishly. There is no paralysis of eye-muscles, and the optic nerve-head shows no pathologic changes. The face is not paralyzed, and the tongue is protruded in a straight line, being clean and free from tremor. The teeth seem in perfect condition, so far as may be judged without X-ray examination; likewise the tonsils. Temperature, pulse, and respiration negative. Blood-pressure, 155 systolic, 90 diastolic. There are no signs of disease in any of the viscera—heart, lungs, kidneys, and abdominal organs. The urine, tested for albumin, sugar, casts, and urea, gave normal values. Wassermann on blood and spinal fluid gave negative results; there was no increase of cells or globulin in the fluid.

To briefly recapitulate the symptoms, negative and positive: Our

patient presents none of the usual evidences of tabes, such as Argyll-Robertson pupils, lancinating pains, or sphincter disturbances in bladder or bowel, nor have we discovered any optic nerve changes. Instead, there is muscular weakness and pain of a continuous and dull character in legs and arms, accompanied by extreme tenderness on pressure of the calf-muscles and of the muscles in the upper ex-



Shaded portion: Anæsthesia and analgesia.

Shaded portion: Anæsthesia and analgesia.

trémities; and these same muscles are found flabby, wasted, and paralyzed. This is a classical picture of multiple neuritis; and in view of the admitted history of chronic alcoholism of many years' duration, and the absence of any of the other factors capable of producing a similar disease picture, the case is clearly one of alcoholic multiple neuritis.

CASE II.—The patient is a German working-woman, thirty-eight years old, who complains of pains in the legs and difficulty in walking. The trouble began as an aching in both legs and thighs two weeks before she was admitted into the hospital. At about the same time, and even somewhat previously, there were constant dull pains which were accompanied by stiffness and peculiar drawing sensations in the calf-muscles, becoming mostly worse toward morning. Bowels are somewhat constipated, but the urinary functions are normal, and there is an absence of sharp shooting pains. With the exception of an attack similar to this one about a year previously her general health has always been good. She is the mother of three healthy children; there were no miscarriages, and she positively denies both venereal disease and alcohol. As to the latter, she went into details by stating that not one drop of spirits had passed her lips during the entire past year.

Examination.—As viewed in diffuse daylight and with an electric lamp, the pupils appear small and respond sluggishly both to light and in accommodation—a phenomenon which must be considered pathologic. The knee and heel reflexes are practically absent, though faint contractions can still be observed in the adductor muscles of the right side when the corresponding patellar tendon is struck with a percussion hammer. Muscle power is not diminished in the lower extremities, but the feet and legs perspire freely. Similarly, there is little or no diminution of strength in the upper extremities. However, subjective sensation is markedly disturbed in both upper and lower extremities: the slightest touch or light pressure on the prominent muscles of both upper and lower extremities elicits violent reactions of pain and discomfort. In contrast with these findings, objective sensory tests—as applied with a camel's-hair brush for touch and with a needle-point for pain—fail to elicit anything abnormal. To compensate for this we discovered a symptom not so common in this disease, namely, extreme ataxia, both static and dynamic; that is, when patient stands with eyes closed and feet approximated there occurs marked swaying of the entire body, and when the patient attempts to walk there is lateral swaying much like that of a drunken person.

Briefly, the patient complains of pains, paræsthesiæ, sweating, and ataxia of station and gait. The examination reveals sluggish

pupils and absent tendon reflexes, but there is no anaesthesia, analgesia, nor loss of deep sensibility.

Are these findings sufficient for a diagnosis? This question can be answered in the affirmative provided certain points are cleared up. First, as to the sluggish pupils present in our case. How can we reconcile a sluggish pupil with the diagnosis of multiple neuritis? The answer is that there are cases of chronic alcoholism with sluggish pupils which become normal after the patient's recovery. The next question is: Can we have multiple neuritis without muscular wasting and without objectively demonstrable loss of power? We now know that there is a type of the disease in which the sensory portion of the mixed spinal nerves is principally affected which has to do with the maintenance of coördination and the production of the tendon reflex, while the motor division of the nerve escapes. So closely do these unusual forms of multiple neuritis resemble tabes that some authors have described them under the special name of pseudo-tabes peripherica. Of course, this would also explain the marked ataxia in our patient.

There are other points which enable us to reach a positive diagnosis in this case. One of these is the discovery of an alcoholic history, in spite of the patient's statement that not a drop has passed her lips within a year. Her relatives informed us that the patient was "tending bar" in her own saloon until a few weeks ago, and that she drank "like a fish." The mere fact of the patient having been in the saloon business would prejudice us in the assumption that she drank immoderately of alcoholics. It is well known that persons engaged in the retail liquor traffic, especially women, derive much of their income by treating and being treated by customers, with the inevitable result of overindulgence. We have thus learned that our patient has habitually poisoned her system with alcohol, supplying us with another missing link in the diagnosis.

A point of negative value, in so far as it excludes the diagnosis of the only other disease for which this case might be mistaken, namely, tabes, is the absence of other symptoms of tabes and the negative Wassermann reactions on blood and spinal fluid. And, last but not least, the mental picture presented by the patient when I first examined her, and still present to a degree, adds a final prop to the diagnosis. During several conversations with me she appeared

completely disoriented as regards time, place, and persons. For instance, when asked whether she knows who I am, she invariably answered that she never saw me before, though she had previously spoken to me on three different occasions. Asked if she was aware of the name of the place in which she found herself, she answered that she was in a hotel. The inquiry as to time she answered that it was morning when it was afternoon. The symptom of fabrication was also quite marked. For example, the question as to what she did yesterday she answered by detailing visits she made, some on the north, others on the south side, when, as a matter of fact, she had not been out of bed for the past three or four weeks. This mental syndrome—consisting for the most part in a confusion as to time, place, and person, added to which there is a tendency to confabulate so as to fill up memory blanks—was first described by Korsakoff, a Russian physician with much experience among patients afflicted with alcoholic multiple neuritis. The disease, or rather syndrome, is called “Korsakoff’s psychosis,” and is commonly associated with alcoholic multiple neuritis, though it may be found in other forms of multiple neuritis and in other conditions not caused by alcohol. The great majority of cases, however, appear in combination with this variety of multiple neuritis.

CASE III.—The patient is a young Englishman, twenty years old, who complains of weakness in the legs and inability to walk. The trouble began about seven weeks ago with a feeling of weakness and numbness in both legs and arms. During his work he frequently noticed that his arms and hands would tire easily, and he would then be compelled to take a short rest. On his way home from work his legs would give way, compelling him to make several stops on the road. This condition gradually grew worse, so that later he needed assistance to get about, but eventually the weakness became a real paralysis. Simultaneously with the weakness there appeared a marked shrinking or wasting of the muscles, combined with sharp pains coming on spontaneously and at irregular intervals. In addition there was a more or less constant dull pain affecting principally the muscles at the periphery of the body. These nervous phenomena were preceded by marked gastro-intestinal symptoms—diarrhoea and vomiting, also constipation. There was slight fever and temporary urinary disorder; patient could not empty the bladder.

Examination.—Beginning from above downward we examine the pupils, which react normally to light and in accommodation. The ophthalmoscope reveals normal fundi. There are no facial asymmetries—the patient can wrinkle his brow, close the eyes firmly, raise the upper lip, protrude the tongue in a straight line, and move the jaw from side to side against resistance. Similarly, the muscles of the shoulder girdle appear quite normal. A different picture is encountered, however, when the peripheral parts are examined: the arms, forearms, and hands are wasted and flabby. The wasting is especially marked in the small muscles of the hands, including the *dorsum*. The palm of the hands is flattened, hollowed out, and the other muscles supplied by the median and ulnar nerves have practically disappeared. The paralysis becomes the more noticeable when the patient attempts to use his hands in the removal of shoes and stockings, a task which he can not accomplish. Unlike the first case demonstrated as alcoholic multiple neuritis, the weakness is not shown merely in the existence of wrist-drop, but also by the patient's inability to use the *small muscles* of the hand. When I place my finger between his thumb and index-finger and the patient makes an effort to press on my finger, no power is developed. On account of paralysis of lumbrical and interossei muscles, abduction and adduction of the fingers is not possible. Wrist-extension is abolished. In fact, there is extreme weakness amounting to paralysis in all of the peripheral muscles and nerves of the upper extremities.

The trunk muscles having escaped, we turn our attention to the lower extremities. The thighs are but slightly thinned, but the legs appear almost without muscle. Flexion and extension of the thighs are weak, but there is no distinct paralysis. However, when patient attempts to dorsi-flex his foot he fails completely; indeed, he demonstrates the existence of genuine foot-drop, combined with flexor paralysis.

There is, then, a generalized wasting of muscles in upper and lower extremities, most marked in the forearms, hands, and fingers, and in the legs, feet, and toes, which is increasingly severe as the periphery of the body is reached.

Reflexes.—The triceps jerks are still elicitable though reduced, but the wrist-jerks are absent on both sides. Likewise, the patellar

reflexes and Achilles response can not be obtained even with reinforcement.

Sensation.—You may have noticed that while I was examining the patient's reflexes the slight manipulation of the extremities required was sufficient to call forth many expressions of pain: he winced and cried out aloud and seemed to be suffering intensely. All of the manifestations of pain became more accentuated when my hand pressed his calf-muscles. Not only does pain occur on pressure of the muscles, but it also appears in spontaneous attacks without any external manipulations being applied—evidently the result of irritation in the sensory portion of the nerves produced by the inflammatory process.

Objective sensory findings are not as well marked as in our first case. Here there is considerable reduction of sensation, so-called hypæsthesia, instead of complete anæsthesia, conforming, however, to the so-called glove-and-stocking variety of anæsthesia.

A summary of the important findings in this case yields wasting and paralysis in the skeletal muscles of the body, principally the periphery; loss of the deep reflexes in the same parts as the paralysis, combined with the subjective sensory disorder of hypersensitiveness on pressure and hypæsthesia to touch and pin-prick. Negatively, the viscera, including the central nervous system, are free from disease. An investigation of the body fluids, such as blood, urine, and spinal fluid, excludes the existence of other diseases as well as the possibility of specific infection. Both positive and negative findings establish the diagnosis of multiple neuritis.

Having made the diagnosis, it is necessary to determine the etiology of the disease. In our first two cases the cause was found to be the common one for most cases of polyneuritis; namely, alcohol. In this case we have neither a history nor signs of alcoholism; evidences of lead intoxication are wanting, and the urine examination failed to reveal albumin or sugar—thus excluding alcohol, lead, nephritis, and diabetes as possible causes of the neuritis. Preceding the development of the symptoms of the disease proper there has been no infection such as typhoid, diphtheria, or influenza, which makes it fairly certain that we are not dealing with a type of multiple neuritis caused by the toxins of an infectious disease, the so-called toxic or post-infectious form of multiple neuritis. From the history you may

recall the statement that before symptoms of multiple neuritis had appeared there were gastro-intestinal disturbances—vomiting, diarrhoea, and some fever. This symptom-complex drew my attention to the possibility of arsenical poisoning as the probable cause of the neuritis. But how did the arsenic in quantities sufficient to produce poisoning enter the economy? In order to find, if possible, a satisfactory answer to this question, I again scrutinized the facts as to the patient's occupation prior to the beginning of his disease and found the following: Having worked as a farmer last summer, one of the patient's duties was to place a preparation of unknown content, presumably arsenic, in the potato fields. While scattering the arsenical powder over the fields during a period of two weeks, sufficient of the preparation must have entered his own system to produce arsenical poisoning. This, in my opinion, completes the chain of evidence in favor of the diagnosis of arsenical multiple neuritis, which developed within a short time after the acute symptoms of arsenical poisoning had subsided. The finding of the real cause of this neuritis is extremely instructive, for it emphasizes the importance of looking beneath the surface of things. Had we been satisfied with the history as first given by the patient, we might have missed the diagnosis as to the etiology. And, after all, rational therapy must be based entirely on etiology.

CASE IV.—The patient, a married woman, twenty-four years old, entered the hospital for the second time about two weeks ago. During her first stay at the hospital she remained three days, and was sent home somewhat improved, whence she returned seven weeks later. She now complains of weakness and peculiar sensory disturbances in both legs and arms.

Examination.—This may be brief, because of lack of time and to avoid repetition. Beginning at the periphery of the body, I find the toes completely paralyzed and placed at an obtuse angle to the leg. Slight manipulation gives rise to much pain. When attempting to practise dorsal flexion she succeeds only in bending the knee to an insignificant angle, and the foot assumes a dropped position, becoming a so-called "foot-drop." Repeated attempts by the patient to raise the legs are successful only in lifting it to a height of one inch above the bed. In examining the upper extremities we find that the conditions are not identical. Here there is no marked

difference from the normal in the appearance of extensors and flexors of arm and forearm, and the patient has the power to elevate the arms and forearms. A mere glance at the hand muscles, however, reveals generalized muscular wasting—the palm is hollowed out, the thenar and hypothenar eminences have practically disappeared, and the back of the hand presents a series of gutters where there should be muscles. There is no power left to grasp an object.

Reflexes.—The reflexes are absent in the knees and heels and also in the plantar surfaces of both feet. In the upper extremities the triceps and wrist-jerks are normal. Pupils respond to light and in accommodation.

Sensation.—While there is no complaint of spontaneous pains appearing in the lower or upper extremities, slight pressure over the calf-muscles causes much suffering. The nerve-trunks are tender on pressure. Objective tests by means of pin-prick bring out the phenomenon of “delayed conduction of sensation” studied in Case I.

Absence of symptoms referable to brain, spinal cord, and cranial nerves, negative spinal fluid and blood, in a patient who complains of weakness and wasting of muscles in the peripheral parts of the body, with tenderness on pressure over muscles and nerves and absence of the deep reflexes in the affected parts—all of these symptoms make the diagnosis of multiple neuritis certain.

As regards the cause of the multiple neuritis in this patient, whether produced by alcohol, arsenic, lead, or endogenous poisons such as those found in diabetes and nephritis, we are again dependent on an exact history of the case. We are already helped by the knowledge that we are dealing with a painful neuritis, probably of alcohol or arsenical origin. There being no evidence or history of alcoholism, but having learned from the patient that she took some medicine just before the onset of symptoms, our inquiry was naturally directed toward ascertaining the character and kind of medicine taken. She informs us that she mistook a white powder for sugar, with which she wished to sweeten her coffee, but that she now believes that the powder was poison. This story lacked the air of probability, for arsenic packages are usually labelled poison in red ink with a cross-bone symbol prominently printed thereon, and are not found lying about in the neighborhood of the sugar container. Frankly stating our disbelief in her story brought out the truth; namely, that she

had taken rat poison with suicidal intent, and that almost immediately after she had swallowed the poisonous dose symptoms of gastro-intestinal irritation appeared. We may now recall that when she first entered the hospital attention was paid to the acute condition of her gastro-intestinal tract, the vomiting, diarrhœa, etc., but after some improvement she was taken home. Meanwhile the arsenic continued its pernicious work, thus giving us an opportunity to receive her later as a patient in the nervous wards. In reference to the production of multiple neuritis arsenic differs from alcohol, for, while small quantities of alcohol taken over long periods of time are the usual cause of the neuritis—a single large dose not being sufficient to produce the disease—arsenic may produce neuritis by the ingestion of a single poisonous dose, provided the poison has not been entirely removed from the stomach. The lesson is obvious: thorough washing out of the stomach is an absolute necessity in every case of arsenic poisoning. I must not omit to mention that arsenical neuritis may be produced in the same way as alcoholic neuritis by the ingestion or inhalation of small quantities of arsenic, as demonstrated by Case III, that of the young English farming boy.

CASE V.—The patient is a laborer, thirty-five years old, who entered the hospital because of weakness and inability to use his hands for work. Some weakness in the legs is also present, but this causes no disturbance in function. Six weeks after having worked in a lead factory his arms gradually weakened until they reached their present state of helplessness. We are informed that two weeks before the muscular weakness had been noticed the patient suffered from an attack of severe cramps and stomach pains and that he was constipated.

Examination.—Aside from the disability in the upper extremities, patient appears well, although slightly anæmic. The blood reveals a moderate degree of anæmia by the low hæmoglobin content and some reduction in the number of erythrocytes, 3,800,000. In addition, some basophilic degeneration of erythrocytes has been discovered, a finding characteristic of lead intoxication. Wassermann and spinal fluid were negative, and the urine was free from pathologic ingredients.

In examining the muscular system, the shoulder group of muscles appear normal and there is but slight weakness in the arms. It is

quite different in the forearms and hands; the entire forearm seems weak. There is a considerable loss of power in the extensors of the forearm, though the wasting is not great. The wrist furnishes us with a typical picture of classical wrist-drop. The remarkable thing in connection with this wasting is that the supinator longus stands out prominently among the wasted muscles. This is noted by writers on nervous diseases as almost pathognomonic for lead neuritis. The lower extremities show neither wasting nor weakness in any of the muscles, though patient complains of fatigue after slight exertion. The reflexes are absent in the triceps of both arms, but the supinator reflex is present on both sides. There is no change in the reflexes of the lower extremities.

Sensation seems normal even in the territories affected with muscular weakness and partial paralysis.

The disease is practically a motor disturbance; yet, because of the symmetrical involvement and loss of reflexes, it belongs in the class of multiple neuritis.

What is the etiology? The blood-picture is that often observed in lead neuritis—slight anæmia, with basophilic degeneration of erythrocytes. The paralysis was initiated by some kind of colic, presumably lead colic, and seems almost confined to the motor system, another quality of lead. Why lead should leave out the sensory nerves and attack the motor portion of peripheral nerves nobody has yet been able to satisfactorily explain. A final proof that we are dealing with a lead neuritis is the appearance of the patient's gums, mention of which I have reserved to the last: at the junction of gums with teeth, and apparently buried in the gum-tissues, there are bluish particles of lead dust which give us the picture of the classic lead line. This is a typical example of lead neuritis of the wrist-drop type, though there exists another but rarer form of lead neuritis, the so-called upper-arm type, in which the arm-muscles—deltoid, biceps, brachialis anticus, and supinator longus—are the only ones affected.

CASE VI.—The patient I now wish to demonstrate is fifty-five years old, a salesman by occupation and the father of four healthy children. He complains of certain peculiar sensations which he experiences in the legs and arms, especially in the legs. He describes his sensations as a feeling of millions of ants crawling up and down his legs and arms. Having been a clothing salesman all his life, he

was never exposed to the fumes and dust of lead and other noxious materials. He is not alcoholic and has escaped venereal infection.

Examination.—The patient appears well nourished; in fact, he may be considered slightly adipose. His gait is uncertain, but this is seemingly the result of discomfort and ataxia rather than of weakness. The upper extremities show no wasting, but the triceps and wrist reflexes are absent. The strength, as tested by the dynamometer, shows some reduction in both hands and no wrist-drop. The lower extremities are not wasted, but both knee and Achilles jerks are markedly reduced, obtainable only with Jendrassik's method of reinforcement. When the patient is made to stand with eyes closed and feet approximated there is considerable swaying of his body, the so-called Romberg sign. There is slight uncertainty in his gait. The cranial nerves show no anomalies; the pupils respond to light and in accommodation, and there are no optic nerve changes. The blood and spinal fluid are negative for Wassermann, and there is no lymphocytosis nor globulin increase.

Essentially, the positive findings are certain paræsthesias in the upper and lower extremities, reduction or loss of deep reflexes, and marked ataxia. Motor weakness is absent.

The diagnosis of this case can be made by excluding syphilis and other organic nervous diseases, because of the absence of symptoms belonging to such diseases. However, the presence of sensory disturbances in the form of paræsthesia and ataxia, as well as a loss of reflexes limited to peripheral parts of the body, makes the diagnosis of multiple or peripheral neuritis quite plausible.

Let us seek out the cause of this multiple neuritis. Having excluded alcohol, arsenic, lead, and other exogenous poisons, we may direct our attention to the possibility of an endogenous poison producing the symptoms. Which are the principal poisons within the body capable of producing multiple neuritis? The first place belongs to diabetes; then nephritis and intestinal auto-intoxication must be mentioned. These considerations lead us at once to make a thorough inquiry into the condition of our patient's urine. Several examinations made of the urine have indeed shown the presence of a small quantity of sugar constantly present. We are now in a position to complete our diagnosis; namely, the case is one of diabetic multiple neuritis. Cases of this type are not at all uncommon, but are seldom

recognized by those in a position to benefit these patients, for of all forms of multiple neuritis this one can be successfully attacked by proper attention to the underlying cause; namely, diabetes.

CASE VII.—The patient is a young man of about twenty-five who complains of uncertain gait; he staggers in walking to a degree that he is unable to get about unless he leans on some one and uses a cane at the same time. This difficulty developed about six weeks after having passed an acute illness, which the patient thinks must have been the grip, because he was feverish, suffered from sore throat, and was generally indisposed for a few days. Four weeks after the acute illness had subsided he noticed that liquid foods had a tendency to regurgitate through his nose and that his voice had changed—had become nasal and indistinct. Within a few days following this he began to experience difficulty with his eyes—he could neither read nor see objects plainly; everything appeared double to him. Two to three weeks after the eye-symptoms had appeared there came this unsteadiness of his gait for which he entered the hospital. His previous health history is good. Being a tailor by occupation, he works long hours, but he states that he never drank to excess, and his indulgence in tobacco is very moderate. There is nothing in his family history which may have predisposed him to the disease, excepting, perhaps, a case of chorea in a younger brother, which may possibly indicate a hereditary tendency to nervous afflictions.

Examination.—The gait is a good example of ataxia as seen in its classical form in tabes dorsalis. In our patient, to the ataxia there is added muscular weakness, as evidenced by a slight foot-drop. The eyes show marked external strabismus, more on the right side than the left; that is, the eyes are turned out, because the external rectus muscles are having full sway in the absence of the proper functioning of the internal recti which are paralyzed. There is also a tendency to ptosis; that is, there is a partial drooping of both upper lids. Of the internal eye-muscles only the muscle of accommodation appears to be involved. The optic nerve is unaffected, likewise the other cranial nerves. The hands are weak but not paralyzed. The lower extremities are more ataxic than paretic, and peculiar perverted sensations are experienced in them—a feeling of ants crawling over the skin and a sensation of numbness.

The deep reflexes are absent in both upper and lower extremities.

The sensory examination reveals some hypæsthesia in forearms and legs as well as in the feet. There is also excessive sweating of the plantar surfaces. Blood and spinal fluid tested for Wassermann were negative. A chemical and microscopic examination of the urine yields nothing pathologic.

This is another case of peripheral or multiple neuritis, because there is involvement of peripheral nerve territories, loss of reflexes, and sensory disturbances corresponding to the same regions, with nothing to account for these symptoms in the central nervous system. When we recall the fact that the patient had passed through some acute illness several weeks before the advent of symptoms, which illness was accompanied by fever and sore throat, we may well assume that the condition is one of multiple neuritis, caused in all probability by the poison left in the system from the acute infection, and that the infection was a mild case of diphtheria. To judge from the number of such cases—that is, those giving us the syndrome of diphtheritic polyneuritis—many cases of mild diphtheria have not been recognized, for the patients are surprised when informed that they had suffered from that disease before their nervous symptoms appeared.

We may now review some of the general features of this interesting disease, for of all organic diseases of the nervous system multiple neuritis offers the best prognosis and is most amenable to therapy.

SYMPTOMS

1. *Sensation*.—In almost every case of multiple neuritis, from whatever cause, some form of sensory disorder appears early and remains late. The disturbed feeling may vary from a mere numbness, pricking, tingling, or formication to the most excruciating pain described as tearing, boring, stabbing, stretching, or burning. Usually dull, the pain is more or less constant, worse at night, but it may be interrupted by paroxysms of severe pain. A form of sensory manifestation appearing early in multiple neuritis is the so-called hyperæsthesia; that is, a light touch with either pin or camel's-hair brush gives rise to an excess of feeling, in some instances amounting to pain. This hyperæsthesia is usually temporary in duration and soon followed by relative or complete anæsthesia, when there will be either decided reduction or complete loss of the perception of touch, pain, and temperature. A delay in the transmission of painful impres-

sions, so-called "delayed conduction of sensation," is considered the equivalent of anæsthesia. Deep sensibility from muscles and joints may be equally affected with the superficial sensation, which accounts for those cases of multiple neuritis in which coördination and ataxia are prominent symptoms. The sign of great diagnostic value is tenderness elicited upon pressure of muscles and nerves. When, in addition, superficial anæsthesia is associated with the deep-seated pain on pressure, the diagnosis of neuritis becomes almost a certainty. Extreme sweatings and hyperæsthesia of the plantar surfaces of the feet may constitute the only symptoms of alcoholic multiple neuritis. The sensory disturbances may be limited to single nerve-trunks or they may affect entire limbs, are most aggravated in the peripheral portions, and diminish in intensity as the trunk is approached. Multiple neuritis is a symmetrical affection, and in this regard stands out clearly against other diseases resembling it.

2. *Motility*.—All grades of motor insufficiency may be encountered, from slight weakness to complete paralysis of all four extremities. A common early sign is extreme fatigue upon slight exertion. One who always considered himself strong notices a reduction in his working capacity, and weakness ensues, which he attributes to irrelevant causes. From a mistaken notion that the weakness is a form of nervousness he remains out of bed and attempts to work. Sooner or later he finds himself completely paralyzed and has to seek a hospital. Quite characteristic of the disease is the extensor paralysis in hands and feet; usually the lower extremities are affected before the upper ones, but the reverse may take place. As peripheral neuritis is an affection of the lower motor neuron, rapid wasting appears in the affected muscles, so that often within a very short period from the beginning of the affection the skin is seen to hang from the bone in large folds without any intervening muscle tissue remaining.

Within a week or ten days following the first symptom some form of electrical reaction of degeneration may be discovered in the affected muscles and nerves. In the cases which I presented no electrical examinations were made systematically because of battery trouble, but this method of examination of muscles and nerves is not only valuable in diagnosis but also in the prognosis of an individual case. In testing for the reaction of degeneration the essential points to remember are: First, response to the faradic current is greatly

diminished or lost in both the affected nerve and muscle; second, the contraction by galvanism becomes slow, vermicular, while the normal muscle contracts with lightning-like rapidity.

3. *Reflexes*.—Changes in the reflexes are noted in almost every case of multiple neuritis. Excepting the few cases in which the deep reflexes are temporarily exaggerated, they are always reduced or absent. In many cases the reflex disturbance is an early sign of the disease and should be looked for in every instance.

4. *Ataxia*.—Disturbance or loss of the muscle- and joint-sense may produce ataxia, which may appear as the sole or the most important symptom of multiple neuritis. Many mistakes in diagnosis have been made because of the presence of this symptom. It should be remembered that the cases of so-called neuro-tabes peripherica or pseudo-tabes are not rarely seen in alcohol or arsenical neuritis, but most often in the diabetic form.

5. *Miscellaneous Symptoms*.—Edema and vasomotor changes may develop in the peripheral parts of the body, which changes may be accompanied by profuse and offensive sweats.

Slight rises in the body temperature may exceptionally occur.

The pulse may become accelerated. A pulse of 90 is still considered within normal limits for multiple neuritis. A pulse-rate of 210 to 140 means danger from involvement of the vagus nerve, and usually foreshadows a fatality.

Exceptionally the cranial nerves may be affected in multiple neuritis, especially in the diphtheritic cases.

TREATMENT

When first called to an acute case of multiple neuritis an attempt must be made to shorten the duration of the disease and to make the patient comfortable. Diaphoretics and salicylates, or aspirin and pyramidon, with sodium bromide in ten- to fifteen-grain doses, may be administered every three or four hours. In my experience aspirin has been very helpful early in the disease when administered in ten-grain doses every three hours for a period of three to four days. Locally hot-water bottles may be applied to the affected parts, guarding the patient against burns. The best remedy for the relief of pain is the application of moist heat by means of local packs. If the patient's general condition permits, general hot baths may be tried.

Morphine should be reserved for the cases in which the pain is so intense that there is danger from this cause alone. If given at all it should be used in single large doses to insure the anodyne and hypnotic effects.

Concerning the management of the various types of multiple neuritis, each variety should receive attention according to etiology. For instance, in the toxic varieties we endeavor to search out the poison having caused the disease and then to remove the poison from the patient or the patient from the poison. Alcoholics should not receive a drop of alcohol in any form and under any pretence. Arsenical neuritis can be benefited before the neuritis has developed; that is, when a patient develops the first symptoms of arsenical poisoning we must endeavor to prevent the slow absorption of the poison into the system. But once the condition has fully developed there is no special treatment. The treatment of lead neuritis is largely of the preventive kind; the time to do something is when lead intoxication is first noticed. Then aromatic sulphuric acid should be administered, or the old-fashioned administration of iodides in fifteen-grain doses three times daily should be given a trial. The diabetic variety is treated the same as the other symptoms of diabetes; namely, by regulating the diet, making it practically sugar- and starch-free. The management of post-diphtheritic cases of multiple neuritis requires much care and individual attention. A patient who is attacked by diphtheria should always be considered a candidate for post-diphtheritic neuritis. Attention should be given to the convalescent period of diphtheria: the heart should not be overtaxed and the brain should be spared as well as the nerves. Many a so-called recovered case of diphtheria died suddenly from involvement of the pneumogastric nerve because the advent of multiple neuritis was not heeded by his medical attendant.

There is a general treatment applicable to all forms of multiple neuritis which consists in the prevention of bed-sores and deformities. To prevent the troublesome bed-sores pressure should be removed from the bony portions of the body. This can be done by the use of water pillows and frequent changing of position. Considering the frequency with which contracture deformities follow multiple neuritis, we should plan early to prevent such occurrences by extending the knees and elbows and keeping them in position by means of

proper immobilizing apparatuses or by plaster-of-Paris casts. The troublesome foot-drop can thus be prevented from becoming a permanent contracture; and a cardboard splint applied in time may again render the hand useful after recovery from wrist-drop. One may begin early to guard against deformity by placing a sandbag against the sole of the foot, even when the patient is still suffering from the acute symptoms of the disease.

In the chronic cases strychnia and arsenic may be given in tonic doses. In fact, the routine treatment of all forms of polyneuritis with strychnia sulphate or strychnia nitrate hypodermically or by mouth has given good results and must be acknowledged to be the best single remedy in this disease. Its administration may be begun with one-thirtieth grain, and it can be gradually increased to one-tenth grain three times daily. This treatment can be continued for months with no unpleasant after-effects.

So soon as the pains have subsided—that is, when the condition assumes the chronic stage—we may begin the application of electricity and general mechanotherapy. Massage and electrical treatment, combined with passive flexion and extension, must be systematically practised over long periods of time.

BLOOD-PRESSURE IN PREGNANCY *

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WE have to-day four cases of high-blood-pressure of pregnancy to show you which illustrate somewhat different phases of this subject, hence I wish to take this occasion to call your attention to the significance and importance of careful and frequent observations of the blood-pressure in every case of pregnancy, for it not only gives us early—perhaps the very first—warning of an approaching toxæmia, but enables us to detect other complicating diseases of pregnancy which are accompanied by high blood-pressure.

Furthermore, these observations should be begun early in pregnancy in order that relative changes during gestation may be observed and any complicating disease detected soon enough to institute proper treatment; for example, if the blood-pressure be not taken till full term approaches, one may interpret hypertension as due to toxæmia of pregnancy which may have existed from the beginning, due to nephritis; thus two errors will have been made: one the failure to detect a kidney lesion which should have been dealt with months before, and the other a mistaken diagnosis and treatment of toxæmia of pregnancy because that is the most likely cause of hypertension at full term.

It took many years to sufficiently impress upon the profession the necessity of early and frequent examinations of the urine as a safeguard against toxæmia, and I have seen occasions when I feared that even this well-established fact was not universally accepted.

I sincerely hope that it will not take as long to drive home the equally well-defined truth that blood-pressure observations during pregnancy are just as important as examinations of the urine, and because it frequently antedates the appearance of albumin it might even be considered more important.

It is to further this idea that I present these cases to-day, to—

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gether with an analysis of blood-pressure observations on 524 additional cases.

General attention has been given to blood-pressure only since about 1905, and during pregnancy for even a shorter time.

In the study of blood-pressure in disease consideration is given to the systolic and diastolic pressure and also the pulse-pressure, which is the difference between the two. In pregnancy emphasis has been placed chiefly on the diastolic pressure. In analyzing these 524 cases from this clinic and from private practice I have taken into consideration not only the systolic, but also the diastolic and pulse-pressure as well, to see if the latter two would give us any additional valuable evidence, especially as to the toxæmias, not revealed by the systolic pressure.

NORMAL SYSTOLIC PRESSURE

Newell¹ avers that the normal limits during pregnancy are between 100 and 130; others assert that the upper normal limit may be 135. In our series of 524 cases, in 306, or 60.5 per cent., the systolic pressure ranged from 100 to 130, and about 20 per cent. so little above 130 as to be considered within normal limits, giving us 80 per cent. practically within these bounds.

Irving,² in his most admirable analysis of 5000 cases, found four-fifths, or 80 per cent., had a systolic pressure never below 100 or above 130. Therefore we may safely conclude that a pregnant woman with a systolic pressure above 130, while not necessarily in immediate danger, should be more frequently observed to make certain that the rise above 130 is not the beginning of a continual increase which may mean toxæmia.

In discussing this phase of the subject Starling⁴ said: "I am convinced that during the whole period of normal pregnancy the blood-pressure is normal. Any blood-pressure above 125 mm. would make me suspect that the pregnancy was not quite normal and would put me on the lookout for some degree of toxæmia."

Vogeler⁷ gives the normal ranges of blood-pressure in the non-pregnant according to age as 100 to 150 mm. of mercury; similar conclusions were reached by Libedoff⁸ and Cook and Briggs.⁹

Child-bearing women, all being relatively young, are not affected

by the normal increase with advancing age, therefore our high normal limit of 130 mm. is approximately correct, to say the least.

J. C. Hirst⁶ found "the systolic blood-pressure in 100 non-pregnant women 112 mm., and in pregnancy 118 mm. up to 7 or 7½ months," after which he says "there is a gradual rise, so that in the middle of the last month the average is 124 mm., and when the subsidence of the uterus takes place there is a slight fall."

Blood-pressure usually falls after rupture of the membranes, lasting two or more hours, according to Hirst⁶ from 60 to 90 points, and a like fall after the child is born, also temporary, lasting, as a rule, not more than a half hour. Even in cases of eclampsia with very high blood-pressure there is usually a primary fall, sometimes as much as 100 points, but it soon rises almost or quite as high as before delivery, gradually subsiding after two or three days, and is usually back to normal in ten days or two weeks.

Donjan¹⁶ asserts that "there is hypotension during gestation, hypertension during birth, hypotension post-partum."

LOW BLOOD-PRESSURE

In our series, in 96, or 18.3 per cent., the blood-pressure fell below 100. Irving¹ found 9 per cent. with a low blood-pressure.

Low blood-pressure has no such dire significance as high tension. Women with low pressure do not seem to be more prone to shock than others. However, I have observed that in nearly every case of low tension the woman is below par, and a careful inquiry will usually reveal a cause for the asthenia, such as excessive vomiting, overwork, or nervous strain, and, if the underlying condition is allowed to go uncorrected, that patient may come to her delivery poorly equipped to endure the stress of labor.

Lynch¹² thinks that "comparatively low blood-pressure may be found in pregnancy with no other evidence of abnormality of health." It is a mistake to ignore low blood-pressure in the pregnant, for I believe that it should be our aim to bring every woman to her confinement in the best possible physical condition; she will stand the strain of labor better and will recover more quickly if she be in sound health, but I have seen no convincing evidence in our series that women with low blood-pressures are in particular danger of shock.

Lynch¹² thinks that "through the study of blood-pressure we may have a forewarning as to the possibility of shock following labor not associated or resulting from hemorrhage."

Sterling,² in taking issue with Lynch on this subject, says: "The Crile theory of shock seems to offer the best explanation. Following rapid evacuation of the uterus, there comes a marked lowering of intra-abdominal pressure, and thereby the external mechanical support of the splanchnic vessel walls is diminished; should vasomotor control be inefficient these vessels become enormously distended with blood, and the patient bleeds into her own abdominal vascular system. The resultant anæmia of the medulla completes the familiar picture of surgical shock. It is evident that such a condition presupposes a certain instability of vasomotor equilibrium; but that it can be foretold during pregnancy by low blood-pressure is not borne out by our observations."

The case of Mrs. B. illustrates this. She was a large, strong, healthy woman, but was melancholy and apprehensive, insisting all through her pregnancy that she did not expect to survive. She had hydramnios of large but slow development. At seven months she was threatened with a premature labor, but went to full term. Soon after delivery she went into alarming shock and died. The amount of hemorrhage was not enough to account for the shock, which in all probability was explainable by the "bleeding into her own splanchnic system," as stated by Sterling. No warning was given by a low blood-pressure, her systolic blood-pressure being 122 mm. and diastolic 80.

Many writers have told us that there is a normal rise in blood-pressure as pregnancy advances, and they substantiate their statements by averaging the blood-pressure of their cases at the different months. This may be misleading, so instead of averaging the blood-pressure I have taken the whole number of cases and figured the percentage that had an increase or a decrease of more than five points between the first and last reading, with the following results:

Percentage of cases showing an increase of five points, 30.

Percentage of cases showing a decrease of five points, 21.

Percentage of cases showing neither increase nor decrease of five points, 49.

These figures are very illuminating, for they show that in this series at least the great majority of cases, 70 per cent., do not show an increase, 21 per cent. even showing a decrease.

This method seems much more logical to me than figuring the average blood-pressure at the different months. These figures include only cases with a blood-pressure within the normal limits, and do not include those with hypertension, for it is manifestly illogical to include abnormal cases when we are trying to determine if there be a normal increase of blood-pressure during pregnancy.

DIASTOLIC PRESSURE.

I have been rather interested in the study of these cases to see whether an analysis of the diastolic pressure and pulse-pressure would reveal any evidence not conveyed by observations on systolic pressure alone. Our cases showed the following:

Diastolic Pressure

50 or less,	35, or 66	per cent.
51 to 60,	94, or 18.0	per cent.
61 to 70,	200, or 38.4	per cent.
71 to 85,	137, or 26.2	per cent.
86 to 99,	44, or 8.4	per cent.
100 or more,	14, or 2.6	per cent.

In analyzing these figures as a group and investigating each individual case any constant prognostic evidence added by the diastolic pressure not revealed by the systolic pressure is not in evidence, although in some cases it apparently was a better indication of a dangerous toxæmia than the systolic pressure, but a relatively higher systolic than diastolic pressure occurred more often than the reverse, as is shown by the increased pulse-pressure, which we will discuss later.

Although the series is small, I can not escape the conviction that the diastolic pressure in pregnancy does not, as a rule, tell us more than does the systolic; neither can I escape the impression, insufficient as the evidence is, that an unusually high diastolic pressure, with or without an unusually high systolic pressure, is of prognostic significance, and I believe, furthermore, that the evidence is not

complete unless we take both pressures, not only that we may study their individual significance, but that we may accumulate evidence which, when it has assumed sufficient proportions, may enable us to draw conclusions not now possible; and, again, the taking of both systolic and diastolic pressures necessary to the study of pulse-pressure, which may possibly also help us to solve this vexing question.

The attitude of internists toward diastolic pressure may be expressed by Rowan,¹³ who said: "Systolic pressure is much less important than either diastolic or pulse pressure. Its diagnostic value is practically *nil*. The diastolic pressure is of the greatest importance in diagnosis and prognosis."

Diastolic pressure is probably not as significant in an acute condition like toxæmia of pregnancy as in the chronic disease of the heart and kidneys. Stone¹⁴ emphasizes the diastolic pressure which represents the constant pressure between systoles, and believes it is a better index of peripheral resistance than the systolic pressure.

Janeway,¹⁵ on the other hand, declared that, except in aortic regurgitation, marked bradycardia, and senile pulse, the diastolic pressure is not of great value.

PULSE-PRESSURE

In chronic diseases of the heart and kidneys our medical colleagues have found that the pulse-pressure may have a greater significance than either the systolic or diastolic pressure alone.

I have been unable to find anything in the literature bearing on this phase of the subject during pregnancy, so will have to be content with the results of our series, which, of course, is too small to justify final conclusions; however, the evidence is valuable as far as it goes.

Inasmuch as pulse-pressure is the real force in driving the blood mass to the tissues, we might, perhaps with reason, expect it to be of prognostic value. Pulse-pressure, which is the difference between the systolic and diastolic pressures, is considered normal when it ranges between 30 and 50 mm. of mercury.

"A pulse-pressure under 30 is evidently not enough, as a rule, to send the blood through the numerous channels and force it back to the right side of the heart. On the contrary, a pulse-pressure of

100 or over is compatible not only with health, but also with considerable mental and bodily vigor." ⁸

I have divided the investigation into two groups: first, the pulse-pressure in cases with low systolic pressure, and, second, those with high systolic pressure.

The pulse-pressure in 79 cases with a systolic pressure of 100 mm. or below ranged from 6 to 52, distributed as follows: 37 per cent. had a pulse-pressure below the normal low range of 30 mm., 56 per cent. were within the normal range of 30 to 50 mm., and 7 per cent. above the normal high range of 50 mm.

These figures show that most of the pregnant women with a relatively low systolic pressure have well-balanced circulation, most of them having a normal pulse-pressure; however, many of those with a low pulse-pressure were suffering from conditions which cause debility, such as vomiting and overwork or strain upon the nervous system.

We are more interested in the cases with high systolic pressure, because they are the ones threatening toxæmia, and we want to know if the pulse-pressure will tell us any more than does the systolic and diastolic pressures or add to the evidence they give.

Pulse-pressure in 92 cases with a systolic pressure above 130 ranged from 20 to 106. Three only had a pulse-pressure below 30 mm., 35 from 30 to 50 mm., 49 from 50 to 70, and 6 from 80 to 106. The one case with a pulse-pressure of 20 had a systolic pressure of 140 mm. and a diastolic pressure of 120 mm., and she had no other signs of toxæmia. One with a pulse-pressure of 100 had a systolic pressure of 230 and a diastolic pressure of 130; and the one with a pulse-pressure of 106 had a systolic pressure of 196 and diastolic 90.

These cases would tend to make us think that we might expect a high pulse-pressure in eclampsia. In our series we usually did find the pulse-pressure above normal, but not always very high; it generally was above 70, but we are kept from forming too hasty conclusions when the very next case of convulsions has a normal pulse-pressure of 50, with a systolic pressure of 170 mm. and a diastolic of 120 mm., and, to add to our confusion, another case comes along with the high pulse-pressure of 90, with a systolic pressure of 160 mm. and a diastolic of 70, and entirely unaccompanied by toxic symptoms.

What conclusions, then, can we draw from the cases with high pulse-pressure? None, I fear, that will be of conclusive value; we can only state that usually the pulse-pressure in eclampsia is above 70, but in a small proportion of cases may be lower, even down to normal. So far as this small series is concerned, very little additional evidence is revealed not shown by the systolic and diastolic pressure. However, I do feel that we can not dismiss the question of pulse-pressure as valueless until a much larger series of, say, 10,000 cases has been analyzed.

However, pulse-pressure may be of some value as a therapeutic guide, as Rowan¹³ pointed out when he said: "High pulse-pressure contra-indicates stimulation, and a low pulse-pressure is a sure indication that veratrum will harm the patient."

High tension and albuminuria both are practically constant symptoms of toxæmia of pregnancy; the exceptions are very few. In this fact lies the value of urine examinations and blood-pressure readings; sooner or later they are usually both present.

Eclampsia is in a large proportion of cases a preventable condition if the earliest signs are detected so that treatment may be instituted to prevent a beginning toxæmia from developing into an eclampsia, and, even if the symptoms do not improve and the toxæmia, in spite of treatment, increases, the life of the patient can be saved by a timely interference. Fortunately we do not see as many of the so-called fulminant type of eclampsia since we have been giving more attention to the blood-pressure, for the reason that increasing tension often appears sometimes before albumin in the urine, and treatment can be started much earlier than when we depended on urine examination alone.

I called your attention a little while ago to the desirability of being on your guard whenever the systolic pressure is above 130 mm. of mercury. It is interesting, in this connection, to note that about 3 per cent. of the patients with a systolic blood-pressure between 130 and 140 had toxæmia, showing that even with this low range we may have not only a warning of an approaching toxæmia, but it may actually exist, as proved by other symptoms.

But when we come to the pressures between 140 and 150 the percentage of cases is three times greater, and we are warned of the greatly increasing danger when we learn that more than one-third

of all patients with pressures between 150 and 160 have toxæmia, and we should be truly alarmed if the systolic pressure goes above 160 mm., for one-half of the women with pressures ranging from 160 to 180 are affected with toxæmia, and above 180 practically 100 per cent. have toxæmia.

In 1908 Badger¹⁰ said: "How early in the development of eclampsia the blood-pressure rises I have been unable to determine from the literature. Future observations may show that it is one of the earliest signs." His prophecy has come true.

I have been impressed for a long time by the fact that hypertension frequently appears before albumin could be detected in the urine even in small amounts, but I have not known how often this occurred till I analyzed this series of cases with the following results:

Showing both hypertension and albumin (early), 15 per cent.

Showing albumin with no hypertension, 31 per cent.

Showing hypertension without albumin or before the albumin, 46 per cent.

Showing albumin first (not including those showing albumin without hypertension, *i.e.*, hypertension following later), 8 per cent.

Uncomplicated albuminuria is more frequent than uncomplicated hypertension. "Elevated blood-pressure is, *per se*, a more frequent sign of toxæmia."²

The above figures are quite convincing that hypertension is more apt to be the earlier symptom of toxæmia than is albumin.

Irving² also found this to be true, saying: "An elevated blood-pressure is often the first sign of approaching toxæmia. In the present series (5000 cases) it preceded albuminuria twice as often as it followed it."

There are a few cases of eclampsia which never show any albumin, and likewise there are some which do not have high blood-pressure. When we know the etiology of the toxæmias of pregnancy this unexplainable fact may be cleared up through metabolic studies.

Ewing and Wolf⁵ think that "dangerous toxic states may exist with great metabolic disturbance without albumin in the urine." There probably are different kinds of toxæmia or various not well-understood diseases with high tension which may some day be apparent when we know something more of the etiology. As Faught¹¹ says: "Some women show a persistent high blood-pressure and at no

time present a picture of toxæmia; these should be separated from the pregnant cases showing even a moderately high blood-pressure accompanied by some or all of the familiar signs of toxæmia of pregnancy. The first need careful watching and management, but are not subjects for surgical interference, as are the toxæmia cases with gradually rising blood-pressure."

That convulsions may occur with very low blood-pressure is proved by one of our cases who died in convulsions and had a systolic blood-pressure of only 98 mm. of mercury.

Lynch¹² says eclampsia may occur with low blood-pressure, and cites several cases to prove his contention. We call them eclampsia, but that they are the identical disease of which eclampsia is a symptom I doubt. In the toxæmia of early pregnancy the blood-pressure is not high. In none of our cases of hyperæmesis gravidarum was the systolic blood-pressure ever found above 120 mm. Blood-pressure does not aid us in determining what to do in pernicious vomiting.

TREATMENT OF TOXÆMIA

It has not been the purpose of the clinic to-day to go into any detail of the treatment of toxæmia, but to impress upon you the value of blood-pressure readings; but, inasmuch as the interpretation of these readings must involve therapeutic measures, it will not be out of place to briefly outline the treatment under varying conditions of blood-pressure. When the blood-pressure is above 130 mm. of mercury or albuminuria is present the diet should be salt free, no meat given, large quantities of fluid, and a brisk saline cathartic, and the patient should be seen again in a few days, when, if she has not improved or other signs of toxæmia have appeared, she should be put to bed and the diet still further restricted.

If the systolic pressure is above 140 mm. of mercury the indications are for even more careful observation, and if above 150 the patient should be seen daily, and when between 160 and 180 it always calls for rest in bed and drastic elimination and strictest diet. If in any of these cases improvement fails or the patient grows worse, it is better to terminate the pregnancy than to temporize till forced to do so by the advent of convulsions.

Few women with a pressure continuously above 180 can be safely allowed to go very long without interference. Of course, in nearly all cases elimination, rest in bed, and restricted diet should be employed before any drastic measures are instituted.

It goes without saying that we do not depend alone upon the blood-pressure readings, but take into consideration as well albuminuria and other symptoms of toxæmia.

Illustrative Cases

I will illustrate the value of the fact that high tension may be the first sign of toxæmia by three cases.

CASE I.—Mrs. B. Due February 2, 1917.

November 21, 1916.—Urine normal. Blood-pressure: systolic, 120; diastolic, 80 = 40 pulse-pressure.

January 10, 1917.—Urine normal. Blood-pressure: systolic, 140; diastolic, 92 = 48 pulse-pressure.

January 19, 1917.—Urine normal. Blood-pressure: systolic, 145; diastolic, 88 = 57 pulse-pressure.

January 26, 1917.—Urine normal. Blood-pressure: systolic, 138; diastolic, 88 = 50 pulse-pressure.

February 12, 1917.—Urine normal. Blood-pressure: systolic, 144; diastolic, 94 = 50 pulse-pressure.

February 16, 1917.—Urine normal. Blood-pressure: systolic, 135; diastolic, 85 = 50 pulse-pressure.

February 22, 1917.—Urine normal. Blood-pressure: systolic, 155; diastolic, 98 = 57 pulse-pressure (date of delivery).

March 4, 1917.—Urine normal. Blood-pressure: systolic, 130; diastolic, 80 (six weeks after delivery).

(In this case, as in all of the following, not all of the blood-pressure readings are given, but, to save space, only those which show change.)

Here we have a case starting with a normal blood-pressure, but in the latter part of the seventh month of gestation the systolic pressure was 140 mm. of mercury. Although this is not an alarming tension, she was put on a strict diet, cathartics, and rest, and was seen frequently; the pressure did not go higher, probably due to treatment, till the day of delivery, when it was 155 systolic and

98 diastolic. At no time was there albumin in the urine. Pulse-pressure showed an increase of 17 points.

I am convinced that this woman was toxic, and that marked symptoms of toxæmia, greater tension, and albumin did not develop was due to timely treatment; in other words, we were enabled by the detection of earliest sign of toxæmia—rising blood-pressure—to institute prophylactic treatment which prevented further development of the disease. We know that she started with a normal tension; it increased; the increase stopped, probably by treatment, and gradually became normal after delivery.

CASE II.—Mrs. W., due February 24, 1917, illustrates the sudden appearance of marked toxæmia and also the increased tension as the first sign of danger.

August 25, 1916.—Urine normal. Blood-pressure: systolic, 120; diastolic, 80.

November 20, 1916.—Urine showed pus and trace of albumin. Blood-pressure: systolic, 120; diastolic, 80.

Under the treatment of rest in bed, large quantities of fluid and hexamethylenamine the pus and albumin disappeared.

January 3, 1917.—Urine normal. Blood-pressure: systolic, 120; diastolic, 90 = 30 pulse-pressure.

January 24, 1917.—Urine normal. Blood-pressure: systolic, 162; diastolic, 90 = 52 pulse-pressure.

The patient was very evidently toxic, headache, malaise, etc.; she was placed on strictest diet, put to bed, given large amounts of fluid and large doses of magnesium sulphate.

January 25, 1917.—Albumin +. Blood-pressure: systolic, 162; diastolic, 90 = 52 pulse-pressure.

January 26, 1917.—Albumin ++. Blood-pressure: systolic, 142; diastolic, 88 = 54 pulse-pressure.

January 27, 1917.—Albumin ++. Blood-pressure: systolic, 140; diastolic, 85 = 65 pulse-pressure.

Vision disturbed, headache. Delivered of a premature living baby.

If we had not been making frequent examinations this case, which in a few days developed a marked toxæmia, might have "gotten away from us."

Here is illustrated also the value of daily observations when the systolic pressure is above 160. Pulse-pressure appears here to be of some value, for, although the systolic and diastolic pressures were lower, the pulse-pressure was 11 points higher and she felt worse and the albumin was increased.

February 9, 1917, two weeks after delivery, the blood-pressure was normal: systolic, 120; diastolic, 80.

CASE III.—Mrs. McC. Due June 6, 1916.

April 18, 1916.—Urine normal. Blood-pressure: systolic, 140; diastolic, 90 = 50 pulse-pressure.

April 27, 1916.—Urine normal. Blood-pressure: systolic, 145; diastolic, 90 = 55 pulse-pressure.

May 9, 1916.—Urine normal. Blood-pressure: systolic, 160; diastolic, 98 = 62 pulse-pressure. (Placed on strict diet and put to bed.)

May 20, 1916.—Blood-pressure: systolic, 135; diastolic, 90 = 45 pulse-pressure.

No albumin appeared in her urine until three weeks after the hypertension began; just before delivery she showed a moderate amount of albumin in the urine and entered labor with some hypertension, which gradually increased during labor and was 200 just before delivery. She had two convulsions after delivery, made an uninterrupted recovery, and on July 26, 1916, had a normal blood-pressure: systolic, 120; diastolic, 80.

Here we were enabled to institute treatment three weeks before any albumin appeared in the urine.

CASE IV.—Mrs. J., due February 26, 1917, illustrates the so-called "kidney of pregnancy," with a large amount of albumin, with no hypertension or other signs of toxæmia.

January 20, 1917.—Albumin ++. Specimen sent by mail. Patient ordered to come to the city at once for observation.

January 30, 1917.—Albumin ++. Blood-pressure: systolic, 130; diastolic, 85 = pulse-pressure, 45.

February 16, 1917.—Albumin ++++. Blood-pressure: systolic, 125; diastolic, 90 = pulse-pressure, 35.

February 22, 1917.—Albumin ++. Blood-pressure: systolic, 130; diastolic, 85 = pulse-pressure, 45.

February 22, 1917, delivered, albumin rapidly decreased and disappeared entirely two weeks after delivery.

This patient well illustrates a class of cases with albumin who have no other signs of toxæmia and the blood-pressure readings help us to differentiate them, as a rule, but once in a while we may meet convulsions with very low blood-pressure, as illustrated by the next case.

CASE V.—Mrs. J. Due April 6, 1914. Admitted to the University Hospital, April 19, 1914, in convulsions. Systolic pressure, 98 mm. of mercury; diastolic pressure not taken; albumin, ++++; hyaline and granular casts. She died April 21, 1914. Although we had no time to make a complete study of this case, it probably was one of nephritis.

CASE VI.—L. B. A typical case of eclampsia, showing all the signs of toxæmia: headache, dizziness, disturbed vision, cedema of feet and ankles, decreased urine, unequal pupils, albumin, hyaline and granular casts, rapid pulse, convulsions, etc. Admitted to the University Hospital.

October 12, 1916, 12 M.—Blood-pressure: systolic, 216; diastolic, 150 = 66 pulse-pressure.

October 12, 1916, 3:30 P.M.—Blood-pressure: systolic, 224; diastolic, 148 = 76 pulse-pressure.

October 12, 1916, 4:30 P.M.—Blood-pressure: (veratrum viride, minims v given).

October 12, 1916, 5 P.M.—Blood-pressure: systolic, 120; diastolic, 70 = 50 pulse-pressure.

October 12, 1916, 10 P.M.—Blood-pressure: systolic, 130; diastolic, 80 = 50 pulse-pressure.

October 13, 1916, 12:10 A.M.—Blood-pressure: systolic, 200; diastolic, 110 = 90 pulse-pressure (in labor).

October 13, 1916, 3 A.M.—Blood-pressure: systolic, 230; diastolic, 130 = 100 pulse-pressure.

October 13, 1916, 4:15 A.M.—Blood-pressure: systolic, 230; diastolic, 140 = 90 pulse-pressure.

October 13, 1916, 5:50 A.M.—Blood-pressure: systolic, 220; diastolic, 120 = 100 pulse-pressure.

October 13, 1916, 9:15 A.M.—Blood-pressure: systolic, 208; diastolic, 110 = 98 pulse-pressure.

October 13, 1916, 12:30 P.M.—Blood-pressure: systolic, 200; diastolic, 120.

October 13, 1916, 5:45 P.M.—Delivered.

October 14, 1916.—Blood-pressure: systolic, 128; diastolic, 75.

October 17, 1916.—Blood-pressure: systolic, 209; diastolic, 110 = 99 pulse-pressure.

October 18, 1916.—Blood-pressure: systolic, 210; diastolic, 110 = 100 pulse-pressure.

October 19, 1916.—Blood-pressure: systolic, 200; diastolic, 110 = 90 pulse-pressure.

October 20, 1916.—Blood-pressure: systolic, 160; diastolic, 90 = 70 pulse-pressure.

October 21, 1916.—Blood-pressure: systolic, 156; diastolic, 90 = 66 pulse-pressure (albumin moderate, few hyaline and granular casts).

October 22, 1916.—Blood-pressure: systolic, 160; diastolic, 100 = 60 pulse-pressure.

October 23, 1916.—Blood-pressure: systolic, 165; diastolic, 100 = 65 pulse-pressure.

October 24, 1916.—Blood-pressure: systolic, 160; diastolic, 90 = 70 pulse-pressure.

October 25, 1916.—Blood-pressure: systolic, 165; diastolic, 100 = 65 pulse-pressure.

October 28, 1916.—Blood-pressure: systolic, 162; diastolic, 94 = 68 pulse-pressure.

Albumin trace, a few hyaline casts. Patient allowed to leave hospital to report to dispensary for further observation. Comment is hardly necessary; even the abbreviated record speaks for itself. This case shows one of the highest pulse-pressures seen in the series, viz., 100, and appears to be of some prognostic value.

CASE VII.—E. D.—Illustrates the fact that convulsions may occur after delivery and even though the blood-pressure has been lowered.

September 11, 1912, 12:55 A.M.—Albumin, + + +; finely granu-

lar and hyaline casts, convulsions, systolic pressure 185 mm. of mercury.

September 11, 1912, 2 A.M.—Delivered by forceps, 2:05 A.M., blood-pressure, 185. 2:30 A.M., blood-pressure, 160. 4:30 A.M., post-partum convulsion. 6:16 A.M., blood-pressure, 155, post-partum convulsions continue with increasing severity. 10:30 A.M., 400 Cc. of blood removed. 10:30 A.M., blood-pressure before blood removed, 145. Blood-pressure after blood removed, 110. Blood-pressure 30 minutes after blood removed, 118. 11:30, blood-pressure, 110, convulsion. 12:30, blood-pressure, 115, no more convulsions. Patient died in coma at 6:50 P.M. Neither delivery nor lowering of blood-pressure affected convulsions or fatal ending. Convulsions occurred with blood-pressure 110.

CASE VIII.—E. J., February 22, 1914. Eclampsia. Abundant albumin and casts. Delivered February 24, 1914. Due March 10, 1914. Had some headache, and during last week persistent swelling of legs; none of hands or face.

February 24, 1914, blood-pressure, 196, dizzy, headache, and while being prepared for induction of labor had a convulsion; cervix manually dilated, followed by high forceps.

February 23, 1914.—Blood-pressure: systolic, 192; diastolic, 118 = 74 pulse-pressure.

February 24, 1914.—Blood-pressure: systolic, 196; diastolic, 128 = 68 pulse-pressure. Before delivery. Albumin, + + + +. Few granular and hyaline casts.

February 24, 1914, after delivery.—Blood-pressure: systolic, 104; diastolic, 38 = 66 pulse-pressure.

February 24, 1914, four hours after delivery.—Blood-pressure: systolic, 130; diastolic, 80 = 50 pulse-pressure.

February 24, 1914, seven hours after delivery.—Blood-pressure: systolic, 150; diastolic, 86 = 64 pulse-pressure.

February 24, 1914, eleven hours after delivery.—Blood-pressure: systolic, 160; diastolic, 94 = 66 pulse-pressure. Veratrum given when blood-pressure dropped to: systolic, 142; diastolic, 80.

March 25, 1914, 3 A.M.—Blood-pressure: systolic, 164; diastolic, 108. Veratrum given when blood-pressure dropped to: systolic, 150; diastolic, 80.

March 25, 1914, 8:15 A.M.—Blood-pressure: systolic, 156; diastolic, 84.

March 25, 1914, 7 P.M.—Blood-pressure: systolic, 158; diastolic, 90.

March 25, 1914, 9:15 P.M.—Blood-pressure: systolic, 162; diastolic, 110.

March 25, 1914, 10 P.M.—Blood-pressure, systolic, 156; diastolic, 96.

Pulse-pressure not extremely high. Diastolic no more significant than systolic alone.

CASE IX.—Mrs. J. R. (3360). Eclampsia. Delivered August 12, 1913, 2:50 A.M. Convulsions on admission; three nurses to hold her.

August 12, 1913, 12:15 A.M.—Systolic blood-pressure, 140.

August 12, 1913, 4:00 P.M.—Systolic blood-pressure, 120.

August 12, 1913, 10:00 P.M.—Systolic blood-pressure, 100

August 15, 1913.—Systolic blood-pressure, 110.

August 17, 1913.—Systolic blood-pressure, 135.

August 28, 1913.—Trace of albumin.

Illustrates that eclampsia may occur with systolic blood-pressure of only 140 mm. of mercury.

CASE X.—Mrs. T. September 5, 1914, 2:45 A.M.; admitted to University Hospital in labor, with convulsions; blood-pressure: systolic, 148; diastolic, 65; pulse-pressure, 83.

This case illustrates three rather confusing facts, viz.: first, that convulsions occur with only moderate rise in systolic pressure; second, that they occur with no rise of diastolic pressure; and, third, that a high pulse-pressure may occasionally be an indication of danger when the systolic and diastolic would not seem to indicate particular danger. She was delivered uneventfully and recovered, the blood-pressure being normal at discharge, September 21, 1914.

CASE XI.—Mrs. J. H. Due April 16, 1915. Illustrates post-partum eclampsia.

Pregnancy normal till January, 1915, when she began to have headaches, which soon became intense and frequent, and she was

dizzy; April 10 feet began to swell, she was nauseated, and vomited a few times.

She was admitted in labor to the University Hospital, April 4, 1915, 10 P.M. Blood-pressure: systolic, 165; diastolic, 90 = 75 pulse-pressure; albumin very faint trace. She was delivered at 10:23 A.M., twelve hours after admission, having had no convulsions.

At 3:45 P.M., five hours after delivery, she had a convulsion; after venesection of 500 Cc., at 4 P.M., the blood-pressure dropped to 135 mm.; at 7 P.M. the blood-pressure had gone up to systolic, 168; diastolic, 100; pulse-pressure, 68, and at 7:15 P.M. she had another convulsion. No more blood was drawn. April 6, 1915, the systolic pressure was 148 mm.; April 10 it was 134, and April 11 it was 126. Pulse-pressure and diastolic pressure seem to be a little more significant in this case than the systolic pressure.

CASE XII.—A. C. This case adds to our confusion by showing that we may have a high systolic, high diastolic, and high pulse-pressure with no convulsions.

August 21, 1914.—Blood-pressure: systolic, 268; diastolic, 176 = 92 pulse-pressure.

September 1, 1914.—Blood-pressure: systolic, 170; diastolic, 66 = 104 pulse-pressure.

September 1, 1914, 2:22 P.M.—Delivered.

September 1, 1914, 3 P.M.—Blood-pressure: systolic, 196; diastolic, 90 = 106 pulse-pressure.

September 1, 1914, 10 P.M.—Blood-pressure: systolic, 142; diastolic, 88 = 54 pulse-pressure.

September 2, 1914.—Blood-pressure: systolic, 174; diastolic, 90 = 84 pulse-pressure.

September 3, 1914.—Blood-pressure: systolic, 142; diastolic, 94 = 48 pulse-pressure.

September 4, 1914.—Blood-pressure: systolic, 140; diastolic, 86 = 54 pulse-pressure.

September 6, 1914.—Blood-pressure: systolic, 120; diastolic, 86.

Here we have the highest pulse-pressure encountered in the series, a very high systolic pressure and a high diastolic pressure, but no eclampsia.

CASE XIII.—This case illustrates the fact that the number and severity of the convulsions do not necessarily depend upon the height of the blood-pressure. She had 19 convulsions and her highest systolic pressure was 178 mm., and she continued to have them when the pressure dropped to 155.

CASE XIV.—Mrs. McA. Due November 22, 1916. Illustrates how a patient may be saved from meddlesome interference by knowing that she had a high blood-pressure before conception. She was referred by Dr. R., an internist, who cautioned me about her high tension, and that the most careful investigation had revealed no cause therefor.

August 14, 1916.—Blood-pressure: systolic, 158; diastolic, 80 = 60 pulse-pressure.

August 23, 1916.—Blood-pressure: systolic, 140; diastolic, 84 = 56 pulse-pressure.

September 8, 1916.—Blood-pressure: systolic, 150; diastolic, 90 = 60 pulse-pressure.

September 22, 1916.—Blood-pressure: systolic, 120; diastolic, 80. Only time normal.

October 24, 1916.—Blood-pressure: systolic, 135; diastolic, 82 = 43 (delivered).

November 7, 1916.—Blood-pressure: systolic, 135; diastolic, 85 = 53 pulse-pressure.

January 10, 1917.—Blood-pressure: systolic, 155; diastolic, 90 = 65 pulse-pressure three months after delivery.

March 14, 1917.—Blood-pressure: systolic, 160; diastolic, 90 = 70 pulse-pressure (five months after delivery).

It will be noted that her pressures were even higher after delivery than before, and that her pulse-pressure was never much above normal. The patient seemed always to be in perfect health, and at no times did she have albumin, casts, or any other sign of toxæmia.

CONCLUSIONS

1. The normal systolic blood-pressure in pregnant women is from 100 to 130.
2. The normal diastolic pressure is from 60 to 85.

3. Tension above these figures calls for increased frequency of examinations.

4. The normal pulse-pressure is from 30 to 50.

5. It is not proved that diastolic pressure has a more valuable prognostic value in pregnancy than systolic, but it adds weight to the evidence.

6. Pulse-pressure also gives additional evidence, and apparently is of some value, but further study is necessary to form any conclusions.

7. It is usually stated that there is a gradual increase in blood-pressure as pregnancy advances. We found only 30 per cent. of our normal cases showed an increase and 21 per cent. even showed a decrease.

8. Low blood-pressure is an indication that the woman is below par, but is not a warning that shock may supervene.

9. One pregnant woman in ten has a systolic blood-pressure above 130 and requires special study.

10. High tension is usually an earlier and more reliable sign of toxæmia than albuminuria. Both are very important.

11. An increasing tension is more significant than a moderate high tension, *per se*.

12. About $3\frac{1}{2}$ per cent. of patients with a systolic blood-pressure between 130 and 140 were toxic; 35 per cent. between 150 and 160; 50 per cent. or more above 160, and 100 per cent. above 180.

13. Some cases of high tension may be due to other diseases than toxæmia or to unknown conditions.

14. Eclampsia is more apt to occur with systolic tension above 160 mm., the danger progressively increasing as the pressure rises, but convulsions may occur with moderate or even low blood-pressure.

15. Nearly all cases of toxæmia have both high blood-pressure and albuminuria.

16. Eclampsia is to a large degree a preventable disease if discovered early enough to institute treatment, which brings us to our final and most important conclusion:

17. Early and frequent observations of blood-pressure (together with urine examinations) is the surest method of detecting the toxæmia of pregnancy.

REFERENCES

- ¹ NEWELL: "The Blood-pressure During Pregnancy," *J. A. M. A.*, January 30, 1915, p. 393.
- ² IRVING: "Systolic Blood-pressure in Pregnancy," *Ibid.*, March 25, 1916, p. 935.
- ³ WARFIELD: "The Significance of High Pulse-pressure," *Ibid.*, March 17, 1917, p. 824.
- ⁴ STARLING: *The Lancet*, September 10, 1910, p. 785.
- ⁵ EWING AND WOLF: *Am. Jour. of Obst.*, March, 1907.
- ⁶ J. C. HIRST: *N. Y. Med. Jour.*, June 11, 1910, p. 1204.
- ⁷ VOGELER: *Am. Jour. of Obst.*, 1907, p. 490.
- ⁸ LIBEDOFF: *Centralbl. f. Gyn.*, 1884, p. 1.
- ⁹ COOK AND BRIGGS: *Johns Hopkins Hospital Report*, 1903, ii, p. 451.
- ¹⁰ BADGER: *Boston Med. Jour.*, May 7, 1907, p. 607.
- ¹¹ FAUGHT: *Jour. Am. Med. Assn.*, February 14, 1914, p. 528.
- ¹² LYNCH: *Surg., Gyn. and Obst.*, 1913, vol. xiii, p. 472.
- ¹³ ROWAN: *Jour. Am. Med. Assn.*, March 18, 1916.
- ¹⁴ STONE: *Ibid.*, 1913, vol. ix, p. 1256.
- ¹⁵ JANEWAY: *Am. Jour. Med. So.*, 1906, vol. cxxxi, p. 772.
- ¹⁶ DOUJAN: *Maladies du Cœur*, September 9, 1916, p. 388.

TIBIAL FRACTURE: MULTILOCULAR OVARIAN CYSTOMA: VESICAL CALCULUS: ENCHON- DROMA OF CHEEK

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CASE I.—M. L., a male, aged twenty-two, sustained a fracture of the tibia and was sent to the Louisville Public Hospital, where a plaster-of-Paris dressing was applied. When I came on duty, about four months later, the patient was still unable to walk, no union whatsoever having occurred. Examination showed that the fibula had escaped injury. The X-ray revealed an oblique tibial fracture with considerable overlapping, the site was entirely covered by a huge callus, and there was nearly one inch shortening.

From the conditions present it appeared the only thing to be done was to resort to open operation and wire the fragments together, or adopt some other appropriate step to secure fixation and union. At my clinic the following day the fracture site was freely exposed by incision under general anaesthesia. There was so much overlapping of the fragments, and the amount of callus was so great, that it was necessary to use the chisel rather freely in securing proper bony apposition. There was considerable oozing from the cut surfaces. The ends of the bone were finally accurately approximated by using a chisel handle and forceps.

For holding the fragments in position I employed an apparatus devised by myself about a year ago. It consists of two ordinary gimlets, three and a half inches long, with German silver handles which can be bent in any direction desired, and a clamp with a set screw which is applied to the handles after proper position has been secured. After exposing the fracture site a hole was drilled in each fragment about one inch from the fracture, and one gimlet was screwed into the lower fragment, the other into the upper fragment; the clamp was then applied to the handles and the set screw tightened, thus producing firm fixation. The wound was closed in layers without

drainage, and a plaster-of-Paris dressing applied, with a window sufficiently large to permit of visual inspection whenever desired.

The patient had no discomfort whatever following the operation; röntgenoscopic examination later showed the bones in proper position, and a perfect result was obtained. The gimlets were removed sixteen days after their application; the plaster dressing was allowed to remain six weeks. This simple apparatus holds the bones perfectly firm until the plaster dressing can be applied. The gimlets are easily sterilized, and if there is any difficulty in securing proper alignment either handle can be bent until satisfactory position is obtained.¹

I have under observation another patient with tibial and fibular fracture, the X-ray showing much the same condition which was present in the ununited fracture just mentioned. There is considerable overlapping of the fragments, and the position is unsatisfactory. There is at least half an inch shortening, and, while union might occur in this abnormal attitude, there would, of course, be malposition of the leg with shortening. At my clinic to-morrow I am going to incise the fracture site and apply the gimlet apparatus in the manner just described.

My view is that, at the present day, when so much is expected of the surgeon, when a malpractice suit is often instituted even after the best possible result has been obtained, we are going to resort more and more to the open method of treating fractures, especially those of the long bones. While the gimlets mentioned are easily applied after the fracture site has been exposed, it is best to have the patient in the hospital where the surgeon can observe the result from time to time under the X-ray and thus be assured of the maintenance of proper adjustment. Little risk is incurred in exposing the fracture site; the parts are not touched excepting with sterile instruments, the gloved fingers of the operator not even coming in contact with the wound. By care in asepsis and aseptic technic one is able to expose the line of fracture and correctly adjust the fragments under visual inspection; then with this apparatus firm fixation can be secured.

Where it is necessary to treat a fracture in the patient's home, no apparatus of this kind should be used, and the ordinary methods must

¹ For a more detailed description of the device mentioned, see article by the writer in the *American Journal of Surgery* for April, 1917, in which eight cases are reported successfully treated by means of this apparatus. Illustrations of the device also appear in that paper.

be employed; but where operative steps are undertaken, especially where the patient is able to bear the expense of hospital treatment, and where difficulty has been experienced in securing and maintaining correct apposition, the device described will be found simple and effective. No difficulty whatever has been experienced in screwing the gimlets into the bone after a hole has been drilled. Allen's method of introducing gimlets blindly through the soft tissues can be satisfactorily accomplished only by a surgeon with great experience. He must have the patient on the table under the X-ray to be certain the fragments are in proper apposition, otherwise the method is inapplicable. Where an incision is made exposing the fragments, the certainty of correct apposition is assured. If any form of apparatus is to be used, I believe it wise to make an incision under careful asepsis, and after this has been done the device I have described will greatly simplify the treatment of fractures of the long bones.

CASE II.—Mrs. S. M., aged fifty-five years. I was called by telephone to see this patient, who was in an automobile, awaiting admission to the hospital. She was lying in the back seat and looked as if she had a pillow under her belt; her abdomen was immense, and I immediately thought of ascites or a large ovarian tumor. There appearing no local cause for ascites, I remarked that "we would probably find an ovarian cyst," and was informed this was the diagnosis which had been made by the family physician.

The patient stated that the abdominal enlargement had been gradually progressive for about seven years; that during the last four months the tumor had increased with greater rapidity than before; that, while she had previously suffered only a moderate amount of discomfort, during the last two weeks pain had been severe and it had become extremely difficult for her to assume the recumbent posture.

Examination revealed that she had a loud and distinct aortic systolic murmur, and her heart action was also irregular. Abdominal palpation showed decided fluctuation distributed over a large area, and I had no doubt an enormous collection of fluid would be found when the abdomen was incised. The abdomen was perfectly symmetrical; the umbilicus projected like a small hernia, but no hernia was present. Dulness extended low over both sides, showing that the intestines were crowded out of place in some direction, tympany

being slightly greater on the left than on the right side. No definite tumor could be outlined.

The patient gave the history that a tumor had first been noted in her left side six or seven years ago, and her physician told her then that he thought she had either an ovarian or uterine tumor which should be removed. She told me, among other things, that she was the wife of a blacksmith in the country, that she had been in the habit of helping him in the shop, that the morning before leaving home in the automobile to come to Louisville to be operated upon she had milked four cows, and that it had been her custom to do the milking regularly. It seemed uncertain whether the enlargement of the abdomen might not be due to ascites, although a competent man had made the diagnosis of left-sided ovarian tumor.

The patient was operated upon immediately. The abdomen was sufficiently incised under local anæsthesia to expose the peritoneum, which was then explored, disclosing a firm cystic tumor underneath. A general anæsthetic was then administered and the abdomen opened by a large incision, the cyst was exposed, a trocar introduced, and a large quantity of thick, brownish-red material thus withdrawn, leaving a cavity, which was further cleansed by swabbing with sponges. After more than a half gallon of this fluid had escaped, the trocar was inserted into another cyst and between five and six gallons of fluid evacuated, much of it being lost on the floor. The sac was then removed after separating numerous adhesions involving the omentum and other viscera. A portion of the omentum had to be ligated. The ileum to the extent of about twenty inches was firmly adherent to the tumor and had to be dissected loose. A portion of the mesentery was detached and was sutured into place. No injury was inflicted upon the intestine during the operation. The tumor was composed of several large and small cysts, the solid portion weighing twenty pounds, and the fluid weighed over forty pounds, making the total weight approximately sixty-five pounds.

An interesting feature is that during the operation some of the fluid escaped into the peritoneal cavity, yet, despite this and the fact that the patient lost this immense amount of fluid and solid material, there was no reaction following the operation, her pulse was never above 80 and her temperature was normal. The following day, however, her temperature rose to 101° F., probably due to auto-intoxica-

tion. It is now nine days since the operation was performed; the patient is in a perfectly normal condition, and is talking about returning home.

This case is reported because of the following features: (1) That this woman, crippled for six or seven years by the presence of this large abdominal tumor, had continued working until three hours before the operation was performed; (2) that, despite her rather advanced age and damaged heart, she was successfully operated upon for a serious condition, and has made a prompt, uneventful recovery.

CASE III.—B. F., a male, aged sixty-eight, had been under the care of a competent physician in the state for about six months, and had been treated for what was supposed to be cystitis. The patient was brought to Louisville, and I had no difficulty in making the diagnosis of vesical calculus.

To show that a large vesical stone may sometimes be overlooked even by a careful observer, it is interesting to note that a correct diagnosis was not made in this case, although the calculus had existed for two or more years. The stone weighed four and a half ounces when removed by suprapubic cystotomy.

The patient had suffered considerable discomfort and pain at various times for many months, but had not been confined to his bed, having been able to walk about most of the time, and his general health had remained good. Frequency of micturition and pain during the act were the symptoms upon which the family physician had made the diagnosis of cystitis. The kidneys were shown to be sound by careful examination and the phenolsulphonephthalein test.

It was impossible to get any water to remain in the bladder. Therefore, after making the suprapubic incision and pushing the peritoneum upward out of the way, a sound was introduced, and with this I was able to push the bladder wall well into the wound. A ligature attached to either side made it easy to keep the wound open and the bladder elevated. Forceps were introduced, and, after finding the shortest diameter, the stone was easily removed. I was surprised to find the patient had no prostatic enlargement, which was easily demonstrated after the bladder had been opened.

A large drainage-tube was placed in the bladder with a purse-string suture around it. The bladder wound was carefully closed around the tube, which was left *in situ* for seven days. During that

time not a drop of urine leaked around it, nor was there any leakage into the surrounding tissues, all the urine passing through the tube. At the end of the seventh day the tube was clamped and the patient allowed to retain his urine for three or four hours; he then began voiding. The next day the tube was removed and a piece of adhesive plaster placed over the skin wound. The operation was performed two weeks ago, the wound healed quickly, the patient voids his urine without difficulty, all the symptoms have disappeared, and he will leave for his home in the country to-morrow.

It is a very fortunate termination after the removal of an unusually large bladder stone. It appeared to me that by tightly closing with catgut sutures all the tissue in the prevesical space, after I had placed a purse-string suture around the tube which held it firmly in position, leakage could be avoided and the patient would get well without trouble—which proved to be the case. The greatest danger in cases of this kind is infection of the prevesical space. Fortunately this was avoided; there was no extravasation of urine, the dressing never being soiled.

The most interesting feature about the case is the unusually large size of the stone, which had existed over two years in a man of sixty-eight whose prostate was absolutely negligible and kidneys sound. It is the second largest bladder stone I have ever removed. For some reason, in the course of my surgical experience I have not seen as many vesical calculi as might have been reasonably expected, and those which have come under my observation have been associated with enlarged prostate, excepting in two or three instances in children. In this case the prostate was normal in size and there was no renal disease.

CASE IV.—S. T., a female, aged fifty-five, sought my advice because of a large tumor of the right cheek, which she said was first noticed three years previously. Pain had been a factor from the beginning, but during the last few months it had become much more severe. The tumor bulged into the buccal cavity, and there was a constant dribbling of the mucopurulent material from the patient's mouth. She had become considerably emaciated, due partly to anxiety concerning her condition, partly because of her inability to eat on account of obstruction of the neoplasm, and partly because of the constant pain which disturbed her rest.

Upon examination it appeared that the tumor involved the entire right superior maxillary bone, and its point of origin was thought to be the antrum of Highmore. It looked like an enchondroma, and, as the patient had suffered considerable pain and had lost flesh, malignancy was feared. Careful röntgenoscopic examination revealed nothing.

After due consideration I concluded that this was a case where the superior maxillary bone should be excised—if such an operation is ever justifiable for malignancy—and at the patient's urgent request operation was undertaken. An incision was made along the side of the nose, the lip was separated and the flap then reflected. After this had been accomplished it was found feasible to separate the tumor from the palate bone and remove it, together with the alveolar border of the superior maxillary, without disturbing the remainder of the bone. A preliminary ligature was placed around the carotid artery, but was not tied. The growth had its attachment below the palate in the upper alveolar border of the superior maxillary bone; its structure was firm and cartilaginous-like; the base was small and was well cauterized with thermocautery before the integumentary flap was replaced. The antrum of Highmore was examined and found perfectly smooth; it contained some necrotic tissue, which was removed with a curette. At the time of removal the tumor measured two and a quarter inches transversely and was three and a quarter inches long.

Notwithstanding the appearance of malignancy when the patient came under observation, the tumor was shown by the pathologist to be a simple enchondroma, and the danger of recurrence is slight. Had the true condition been recognized the growth might have been removed without resort to the flap operation; but I had no other expectation than excising the superior maxillary bone, and the incision was made with the view of completing the operative steps as quickly as possible. The ligature around the carotid artery might have been a safeguard had removal of the bone been necessary.

SURGICAL CLINIC OF THE POLYCLINIC HOSPITAL, PHILADELPHIA

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SINCE our report of the Surgical Clinic of the Presbyterian Hospital, which appeared in the June number of *INTERNATIONAL CLINICS* (vol. ii, series 27), we have encountered at the Polyclinic Hospital a number of cases of exceptional interest. From a wealth of clinical material we have selected only those cases that vividly and forcefully elucidate a striking lesson in clinical surgery. To bring out the salient features of each case requires close study and careful, painstaking observation far beyond that usually bestowed in the course of routine hospital work. For the convenience of our readers the cases have been arranged in orderly sequence from head to foot. The initials of the reporter responsible for each case are placed at the end of each case-report.

HÆMATEMESIS DUE TO CONCEALED HEMORRHAGE FROM CORONARY ARTERY OF LIP

CASE I.—M. F., white, male, aged fifty-six, steeplejack, was brought to Polyclinic Hospital by patrol wagon at 12:50 P.M. on December 15, 1916 (Case Record No. 30,258). He was picked up on the street unconscious, and at first no history was obtainable. On admission he was unconscious, and remained so for two hours, or until 2:50 P.M., at which time he was first seen by the reporter, who had no sooner reached his bedside than the patient vomited suddenly four pusbasinfuls of blood, which was not bright red and was mixed with stomach food contents: the total quantity vomited was one quart, approximately. Having recovered consciousness, the patient was now able to state that he had been a lifelong drinker, whereupon the case assumed the appearance of hemorrhage from ruptured œsophageal varix due to cirrhosis. The lower limbs were elevated, the head was lowered, and a hypodermic injection of morphine sulphate, gr. $\frac{1}{2}$, was ordered pending further examination. Hasty examination of abdomen revealed a normal area of liver dullness and no signs of collateral functioning of the accessory portal system of Sappey. It was now noted that blood

¹ Service of Prof. John H. Jopson.

² Service of Prof. George P. Müller.

seemed to be bubbling from the mouth, this phenomenon occurring during expiration and between the intervals of hæmatemesis. There was no external wound of face. Examination of mouth with head mirror and mouth-gag revealed a lacerated wound of inside of left cheek, $\frac{3}{4}$ inch behind angle of mouth. The wound was filled with blood-clots, from the midst of which jets of arterial blood were spurting into the mouth cavity. The clots were brushed away, the bleeding vessel was clamped and ligated with black silk. Pending the fetching of the ligature material and clamp the hemorrhage was controlled by digital pressure upon the facial artery as it crossed the body of the mandible. The wound was closed by a suture of No. 2 chromic gut. The patient now stated that he was very thirsty and drank several glasses of water. He was sent up to ward and put to bed.

Several days later the patient stated that he had cut his cheek during a brawl, in which he was struck upon the chin, a tooth being driven into the cheek. This occurred a week previously and had not been followed by unusual hemorrhage. On the day of admission he was again injured, the tooth again being driven into the same wound.

Interpretation.—The patient was rendered unconscious by a blow upon the chin, which reopened the old wound, this time severing the coronary artery. During the period of unconsciousness the blood from the spurting artery trickled down the pharynx into the stomach. On recovering consciousness the patient vomited the now old blood.

An almost identical case is described by Erichsen,³ who writes: "If the *lip be cut from within*, by being struck against the teeth, the coronary artery may be divided, the patient swallowing the blood that flows into the mouth. Some years ago a man was brought to the hospital, drunk and much bruised about the face. Shortly after his admission he vomited a large quantity of blood, *which was at first supposed to proceed from some internal injury*; but, on examining his mouth, it was found that the blood came from the coronary artery of the lip, which was divided with the mucous membrane."

Dr. John H. Packard, at the meeting of the Philadelphia Academy of Surgery on November 7, 1881, presented a patient who was brought to the Episcopal Hospital blanched and much prostrated by bleeding from a wound in the left cheek, *about $\frac{3}{4}$ inch from the angle of the mouth*. The man stated that the wound had been inflicted with a penknife about ten days previously, and that two severe hemorrhages had occurred—one on the eighth day, the other

³"The Science and Art of Surgery," 1873, vol. i, 508.

on the evening before admission. Both ends of the divided facial artery were ligated with silk. In this instance the wound was external and readily seen, but the patient, as in my case, likewise suffered a severe secondary hemorrhage. The importance of exploring for concealed hemorrhage stab wounds over the known course of an artery or vein—especially a parietal vessel—has been previously emphasized by the reporter.⁴

An extensive paper could be written with the caption "On Hæmatemesis from Extra-gastric Trauma." Suffice it to say, however, that in this respect the stomach may be regarded as a sewer into which trickle fluids from above, whether food, blood, mucous secretions, or what-not. But the stomach is the "watchdog" of the digestive tube, ejecting noxious substances. Raw blood must be as powerful an emetic as mustard, for it is invariably vomited.

That our case of supposed ruptured œsophageal varix resolved itself so simply was, indeed, a source of gratification to all. (P. G. S., Jr.)

FERNICIOUS (HÆMOLYTIC) ANÆMIA; STREPTOCOCCUS HÆMOLYTICUS OBTAINED FROM CARIOUS TEETH; WASSERMANN WEAKLY POSITIVE: IMPROVEMENT AFTER USE OF SALVARSAN AND AUTOGENOUS VACCINE

CASE II.—J. De N., female, white, aged forty-eight, married, referred by Dr. Thomas Tigani, was admitted to Polyclinic Hospital on May 22, 1917 (Case Record No. 31,338). Discharged August 4, 1917. Chief complaints: General weakness; impairment of locomotion. The patient had been perfectly well up to three years ago. At that time she first noticed general weakness and a generally tired feeling. For the past six weeks the lower limbs have felt heavy and she says she feels as if sensation was impaired from waist to toes. She has had increasing difficulty in walking, and at present can not walk unless supported. A friend stated that the patient "always" looks anæmic. Her appetite is poor: she has lost some weight, but does not know how much. She has night-sweats and suffers from headaches and dizziness and also from burning in the bladder after urination. She has had neither nausea nor vomiting. There is some dyspnoea, but no cough.

The following daily notes were obtained from another hospital, where the patient was treated from December 3, 1916, to February 19, 1917:

January 28, 1917.—Patient was transfused with blood and stood operation well.

January 30.—Shows marked improvement in hæmoglobin. Complains of pain in limbs.

February 2.—Unable to walk without assistance, as she has a spastic gait.

⁴"Stab Wound of the Deep Epigastric Artery," *Annals of Surgery*, April, 1917, 451.

February 6.—Still shows improvement of her blood condition; otherwise no change.

February 12.—Blood shows diminution in hæmoglobin and red cells. Apparently there is no permanent improvement from the last transfusion.

February 19.—Patient discharged from hospital in improved condition. Another note shows that while patient was in the hospital the patellar reflexes were *doubtfully present*.

Childbirths.—The patient is the mother of six children—all living and well. Seven years ago she had *two miscarriages*. Her husband is living and well.

Physical examination shows, as the most striking feature, the pallor and lemon-yellow color of the skin; next, that in spite of this dyscrasia the patient seems to be in a good state of nutrition.

Eyes.—Pupils react to light and to accommodation. Ophthalmoscope shows that the media are clear, but that there is a low grade of neuroretinitis in both eyes: in the right eye the disk is pale, its edges are blurred, and the arteries are distended; in the left eye the temporal side of the disk shows a rather pale area around it and hazy, tortuous vessels. There are no retinal hemorrhages.

Nose and Ears.—Negative.

Mouth.—Some teeth are missing and others are in a state of decay. There is some marginal gingivitis. The tonsils are negative.

Lungs.—Expansion fair and about equal on both sides. Voice sounds, breath sounds, and resonance are about normal.

Heart.—Sounds are of fair quality. There is a systolic (hæmic?) murmur at the apex, which is transmitted upward into the precordium, but not transmitted to the axilla. There is no arrhythmia.

Skiagram of Thorax.—Shows enlarged mediastinal lymph-nodes. There is no evidence of disease of the bony spinal column shown in the plate.

Abdomen.—Negative for tumors or abnormal size of any organ.

Skiagram of Stomach and Digestive Tube (Bismuth Meal).—Negative for neoplasms or other distortions.

Pelvis.—Combined vagino-abdominal and recto-abdominal palpation reveals no disease of uterus or ovaries or tubes. There is no infiltrate in Douglas's cul-de-sac.

Neurologic.—(1) Spinal Cord: The patient complains of pain in both sides of abdomen; *these pains are "girdle" pains*. She can not stand up with her eyes closed without swaying (*positive Romberg's test*). Her legs are very feeble, and she must be supported. She has no incontinence of urine or feces. She has pain all the time when moving about. The dynamometer, when placed in the right hand and squeezed, registers 15; left hand, 30. There is no point of hyperæsthesia along spine; there is no tenderness along any nerve-trunk. The patient feels the prick of a pin-point over the entire abdomen and over both lower limbs. The knee-jerks are increased and equally so; there is no ankle clonus; Babinski's plantar reflex is positive on both sides. Oppenheim's test is negative.

Temperature.—At no time above 99° F.

Pulse and Respiration.—Normal.

Urine.—Flocculent sediment; amber; 1.016; acid; no albumin; no glucose; no casts; few epithelial cells, few leucocytes, few erythrocytes; no crystals. Tests for urobilin and urobilinogen negative.

Fæces.—Negative for intestinal parasites, ova, blood.

Blood.—Hæmoglobin, 58 per cent.; leucocytes, 5800; polymorphonuclears, 58; transitionals, 4; lymphocytes, 33; large mononuclears, 4; eosinophiles, 1. Erythrocytes, 2,260,000; resistance—minimal, 9.34; maximal, 9.22. Color-index, 1.2. Normoblasts, 0; anisocytosis, present; poikilocytes, few.

Wassermann Reaction.—On blood, "doubtfully negative"; on cerebrospinal fluid, negative. Four days after a provocative dose of arsenobenzol the Wassermann reaction on the blood was "*weakly positive*."

Cerebrospinal Fluid.—Colorless; sterile; count shows 149 cells; no albumin; no globulin; sugar present. Lange's colloidal gold curve 222 00000—*suggestive of a luetic zone type of reaction*.

Diagnosis.—Syphilitic hemolytic anemia, with spinal cord involvement of the ataxic paraplegic type and neuroretinitis.

Prognosis.—Patient should undergo considerable improvement, both as to the anemia and the spinal cord involvement, provided the treatment planned for her is carried out.

Treatment.—One intravenous injection of "606" salvarsan; one week later an injection of "914" neosalvarsan; one week after this another injection of the same drug. When the blood Wassermann becomes negative, if the cerebrospinal fluid Wassermann is positive give an intraspinal injection of salvarsanized serum according to the Swift-Ellis technic; also, forced feeding, including milk and eggs; ovoferri continuously. When the arsenic medication is completed, give mixed treatment with mercury and the iodides.

This treatment was carried out to the letter. After the first injection (salvarsan) the patient showed marked improvement in that (1) pains in the abdomen and lower limbs were diminished; (2) patellar and Babinski reflexes were less marked; (3) patient began to use legs where she had no power before; (4) patient felt better and appetite improved.

After the third injection skiagraphic and bacteriologic studies of the teeth were made. Skiagrams showed abscesses at roots of teeth. Under novocaine blocking of the infra-orbital and inferior dental nerves the carious snags revealed by the skiagrams were extracted and cultures were taken from their pus-bathed roots: bacteriologic examination revealed *Streptococcus hæmolyticus*. Dr. John A. Kolmer prepared a vaccine of this streptococcus, each ampoule containing 1 Cc. and 300,000,000 cocci. Doses of this vaccine were given at intervals of one week, one ampoule representing one dose.

As time went on the blood-picture remained practically stationary. Why was blood-transfusion not performed? Because two weeks after the patient had been transfused with blood in the other

hospital the following note was made: "Blood shows diminution in hæmoglobin and red cells. Apparently there is no permanent improvement from the last transfusion." Why was splenectomy not performed? Because the presence of the spinal cord involvement was considered a contra-indication to this procedure. At the most, blood-transfusion and splenectomy are but makeshifts in the treatment of pernicious anæmia, and they must always remain makeshifts, *because they do not strike at the etiologic root of the malady.*

In every case of pernicious—or, as it is preferably called, hæmolytic—anæmia an attempt should be made by close study and observation to ferret out the basic etiologic factor of the blood hæmolysis. In our text case two probable etiologic factors were thus revealed: first, the evidence of an old syphilitic infection; secondly, the isolation of *Streptococcus hæmolyticus* from ancient dental root infections. An excellent review of the entire subject along these lines has been presented by Mix.⁵

At present, over five months from the beginning of treatment and three months from the time of discharge from hospital, the patient is in reasonably good condition, though somewhat handicapped by the obstinate spinal cord lesion. It is unfortunate that the patient could not have been placed under treatment very early in the course of her now three-year-old malady, for the surgeon is all but impotent when confronted with terminal stages of pathology. (P. G. S., Jr.)

COLOSSAL DIABETIC CARBUNCLE OF NECK: CRUCIAL INCISION AND UNDERMINING;
CURE

CASE III.—J. R., male, white, aged fifty-one, married, moving-picture operator, referred by Dr. Cesare De Leo, was admitted to Polyclinic Hospital (Case Record No. 31,951) on September 8, 1917.

Present illness.—The lesion is now two weeks old; it started as a "boil" on the back of the neck and gradually increased in size until it reached its present dimensions. There is no previous history of a similar condition.

Physical examination reveals a heavy-set patient beyond middle age. The whole nape of the neck is occupied by a huge, dusky-red carbuncular phlegmon with many orifices. The induration extends from the external occipital protuberance, where it is sharply demarcated from the scalp, downward to the vertebra prominens; laterally it extends from one sternomastoid to the other. Examination of urine reveals the *presence of sugar* (1.7 per cent.): therefore ether is contra-indicated, as it might precipitate acidosis.

⁵ *Medical Clinics of Chicago*, vol. ii, No. 3, November, 1916, p. 509.

Operation.—Nitrous oxide-oxygen anaesthesia. With a large plaster-of-Paris scalpel a transverse incision was made from one edge of the carbuncle to the other; crossing this in the midline, a vertical incision was made from border to border; after cutting through infiltrated tissue for $2\frac{1}{2}$ inches the complexus muscles were exposed, corresponding to the anterior limit (floor) of the carbuncle. The four resulting flaps were undermined so as to form two tiers apiece: one plane of undermining lay between the carbuncle and the underlying uninvolved muscles, and the other plane lay midway between the first and the skin itself, the undermining in every instance being carried to the peripheral limits of the carbuncle. Hemorrhage was controlled by very hot water. All spaces were packed with plain gauze saturated with Dakin's solution, and a wet dressing of the same fluid was applied.

Postoperative Treatment.—Immediately after operation the Allen treatment for diabetes was instituted. Sugar disappeared from the urine within forty-eight hours. In the beginning the wound itself was irrigated with Dakin's solution, which was superseded by the constant saline drip. When clean granulations sprung up dry gauze dressings were applied and changed daily. When the large, crater-like wound had filled up somewhat, adhesive plaster straps were firmly applied directly to the granulating area. These straps were changed at first every day, later every other day. At the present time healing is complete with the exception of an area of healthy granulations the size of a silver dollar, over which epithelialization is advancing with great rapidity under the adhesive plaster compression.

A large field for discussion is opened up by this case. In the first instance, *it is a surgical crime, except in emergencies, to subject a diabetic patient to etherization.* The great danger, of course, lies in the liability of the ether precipitating acidosis and coma in the diabetic. On the other hand, as Payr has stated, no diabetic with dangerous surgical disease should be prevented from being operated upon because he is a diabetic. It is therefore a *matter of great importance routinely to determine the presence or absence of sugar in urine before subjecting any patient to operation (except in emergencies).* This patient was admitted to the hospital for immediate operation, and the reporter made a personal trip to the laboratory to be sure about the sugar proposition before permitting the anaesthetic to be begun: otherwise ether would have been given with perhaps disastrous results. Of course, it is proper to consider every patient with boils or carbuncles a diabetic until proven otherwise. The anaesthetic of choice for diabetics is the nitrous oxide-oxygen sequence, but, as Strouse well says, a diabetic, no matter how well treated, no matter how skilful the surgeon, no matter whether the anaesthesia be local or general, may, despite everything.

develop postoperative coma. The danger is always present, and in operating on a diabetic every possible means should be used to guard the patient against this danger. (For an excellent discussion of the subject of diabetes and surgery see Strouse's article in *Medical Clinics of Chicago*, vol. 2, No. 1, July, 1916, 37.)

The next proposition for discussion is the method of operating for carbuncle. If there is one thing that is axiomatic, it is that *a carbuncle should not be tinkered with, but should be early subjected to a radical operative procedure*. Some surgeons make it a practice to ablate carbuncles just as though dealing with malignant tumors. But this practice is unnecessarily severe, in that it sacrifices an unjustifiable amount of tissue which, if left remaining, would eventually recover itself and undergo *restitutio ad integrum*. Had the reporter ablated the carbuncle in this patient he would have sacrificed large portions of the trapezius and splenius muscles, not to mention skin which would have recovered itself and obviated the necessity of skin-grafting. Nor is total ablation in accord with the surgical pathology of carbuncle. A carbuncle with its pus resembles a honeycomb filled with honey, the main difference being that of the geometric regularity of pattern which obtains in the latter. To get out the honey the comb is sectioned horizontally, opening the cells, and the honey drips out. And so it is with carbuncle: as Warren long ago described and illustrated in his work on "Surgical Pathology," the pus in carbuncle occupies fibro-fatty compartments separated by septa; nature's attempt at cure of a carbuncle is in the direction of providing an outlet to the pus contained in these compartments by opening them up on the skin surface, hence the coarse cribriform surface appearance of a carbuncle left to the *vis medicatrix naturæ*. We are but imitating nature, then, when we undermine the flaps of a carbuncle, opening up the numerous little compartments and giving vent to the pus contained therein. From the nature of things, this can not, however, be accomplished without first making the crucial incision, and in doing this care must be taken to go to the very limits of the carbuncular mass. In our text case, owing to the great depth of the carbuncle, the reporter deemed it wise to undermine the flaps in two tiers. At the close of the operation the patient, seen in profile, looked as though his neck were cut half way through, and inexperienced onlookers rubbed their

eyes in amazement at the optical illusion that lay before them of an almost decapitated man! They forgot, however, that depth in acutely hyperplastic tissues is but a relative matter.

The sacrifice of skin and subcutaneous tissues in the ablation method leaves an unsightly depressed area which is slow to heal up and epithelialize, often requiring skin-grafting. By the crucial incision-undermining method, on the other hand, these tissues are for the most part preserved, although the apices of the skin-flaps might undergo more or less necrosis. After the infection has marched away these skin-flaps fall back and more or less completely cover the wound. The edges of the skin-flaps soon take on active cytogenetic properties, but the new epithelium in its advance is liable to be checked by luxuriant granulations. There are several ways of dealing with such a situation: (a) with curved scissors cut away the exuberant granulations, check hemorrhage with a compress, and apply a drying powder; (b) skin-graft; (c) strap with adhesive plaster. This last method is the best: by its pressure adhesive plaster prevents granulations from becoming exuberant, and underneath the plaster strips epithelialization proceeds with surprising rapidity, so that within a very short time the process is completed. Ordinary zinc oxide adhesive plaster strips of suitable width (usually one-half inch) are firmly applied over the wound in an imbricated manner, quite similar on a small scale to strapping for fracture of a rib. It is not necessary to make an aperture for the escape of the laudable pus, for this not only works its own way out, but is harmless, perhaps even beneficial to the healing process: the even pressure of the strips themselves reduces to a minimum the amount of discharge. To keep the wound as dry as possible calomel may be placed over the granulations before the strips are applied: this is the "calomel-adhesive plaster method," previously described and illustrated in colors by the reporter in these columns.⁶ The straps are changed at first every day, then every other day, and finally twice a week.

A word about the *treatment of furuncles*. There is perhaps no common malady that is so obstinate to the ordinary methods of treatment as boils. It is an old, old truism that the more remedies that

⁶ INTERNATIONAL CLINICS, vol. iii, 23d series.

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have been proposed for the cure of a disease the less effective any one remedy is. He who pins his faith on ointments (ichthyol, etc.) will not get good results. The reporter has threshed this whole subject out repeatedly in previous papers, and space forbids repetition. In 1914 he advocated the use of sulphurous acid.⁷ Increased personal experience with cases of boils, and the reports of physicians and others who have tried this remedy, endorse it as having definite, result-getting merit. The dose is from one to two teaspoonfuls of sulphurous acid disguised by a syrup in a large glass of water one hour after meals and three times a day. Almost invariably the druggist takes exception to this dose, but reference to the Pharmacopœia will show that up to three teaspoonfuls may be given with safety. Almost invariably the physician confuses the remedy with sulphuric acid, and almost invariably he inquires whether the strong or dilute sulphurous acid is used. There is but one form of sulphurous acid—the commercial. It is a much weaker preparation than sulphuric acid: the sulphur is in loose combination and rapidly gets into the system. It promotes diuresis very markedly.

But sulphurous acid should not alone be relied upon to cure boils, although it will do so in many cases. Usually the patients have overindulged in sweets—candy, pastry, etc. Therefore reduce the intake of sugar and fried foods. No less important is the local treatment. Cleanse the skin frequently with benzine, followed by alcohol. As to the boil itself, if it is minute (one or two days old) freeze it with ethyl chloride spray until it is frozen quite hard, take a cataract knife and cut out a little square block of tissue with the minute boil in the centre; thus the lesion is aborted by removing the entire pathology at an early stage when but little tissue need be sacrificed. Not more than $\frac{1}{8}$ inch square should be removed by this method. If the boil is larger because older (third or fourth day), hard, and painful, much relief is obtained, not by making a crucial incision at this stage, which is a mistake, but by lifting the cap off the boil and applying a suction cup until a big welt is raised; the pain-producing tension is diminished by the resulting outflow of serum. Repeat this measure frequently until the boil begins to soften: after the core has been expelled the little

⁷ See *New York Med. Jour.*, 1914.

cavity left remaining soon heals up. The best dressing during the hard, painful stage is to apply, after each cupping, gauze wet with Wright's solution. After the core has been evacuated, use dry gauze and later strap with adhesive plaster. (P. G. S., Jr.)

FETAL ADENOMATOUS GOITRE: RESECTION UNDER HYOSCINE-MORPHINE-NERVE-BLOCKING ANÆSTHESIA

CASE IV.—L. W., female, white, aged forty-six, married, referred by Dr. Thomas Tiganí, was admitted to Polyclinic Hospital (Case Record No. 31,901) on August 26, 1917. Discharged September 10, 1917.

Present Illness.—The patient complains of general weakness, rapid heart action, dyspnea, and "stomach trouble." She has not been well since the birth of her first child, twenty-five years ago. She states that she has had a lump in her neck since babyhood. Her bowels are sluggish.

Physical examination reveals enlargement of the right lobe of the thyroid gland to the size of a large lemon. There are several accessory tumors in relation with the upper pole of this lobe. The enlargement is neither hard nor fixed. There is no exophthalmos. Tremor of the outstretched fingers is present. The pulse is irregular and rapid and shows on the chart wide fluctuations in rapidity in the course of twenty-four hours. On auscultation the heart-beats are strong; there is no marked myocarditis, nor is there any murmur.

The abdomen is pendulous, and skiagram shows ptosis of the viscera, the fundus of the stomach being stretched out until it forms a suspensory ligament: there is no delay in the emptying of the organ.

Interpretation.—While in some respects this clinical syndrome could be attributed to Glenard's disease, yet it is believed that the patient's illness is the result of toxic absorption from a degeneration focus in the goitre. Owing to the dubious strength of the heart general (ether) anæsthesia is contra-indicated.

Mode of Anæsthesia.—Two hours previous to operation the patient was given a hypodermic of hyoscine hydrobromate, gr. 1/200, and morphine sulphate, gr. 1/6. One hour later this same dose was repeated. At the time of operation hyoscine, gr. 1/400, was given. *Local Anæsthesia.*—A two per cent. solution of novocaine was employed. The superficial cervical nerves on both sides were blocked at the middle of the posterior border of the sternomastoid muscle, just above the point of crossing of the external jugular vein. In addition, while waiting for the nerve-blocking to take effect, the projected line of incision was infiltrated intradermally. The superficial fascia and platysma were infiltrated through puncture holes in this incision $1\frac{1}{2}$ inches apart, twenty drops being injected through each hole.

Operation.—The technic of removal did not differ from the usual resection operation. The interesting features were that over toward the isthmus the goitre compressed the trachea, accounting for the dyspnea; and that on sectioning the goitre after removal degeneration areas of softening were found, toxic absorption from which doubtless accounted for the patient's general weakness, tachycardia, and "stomach trouble." Histopathologic examination by Dr. John A. Kolmer revealed no evidence of malignant degeneration. While the patient was coming out of the influence of the anæsthesia drugs it was noted

that her voice was as strong as before operation, showing that the inferior laryngeal nerve had escaped injury.

Postoperative History.—The wound healed *per primam* throughout. At first there was moderate febrile (101° F.) reaction, which is not unusual in these cases, and the pulse continued rapid (124) and showed wide fluctuations. The fever soon disappeared, however, and the pulse gradually returned to normal, aided by the administration of the infusion of digitalis. When last seen the patient stated that she was entirely relieved of her symptoms and felt better than she had felt for years.

This case is presented chiefly to emphasize the value of this method of anæsthesia, the technic of which was practically that described by F. H. Lahey, of Boston.⁸ The reporter did not give the dose of veronal the evening before operation, as recommended by Lahey, because this drug is not only an unreliable somnifacient, but it is often followed by a "hang-over," which is more or less disturbing to a nervous patient the next morning. To Lahey's technic the reporter added nerve-blocking of the cervical plexus—an ideal method for operations upon the front of the neck, and one that is very easy to execute. Lahey's series of eighty-three cases without a death speaks well for this method, although Judd, of the Mayo Clinic, prefers the combination of ether with local anæsthesia, believing the addition of ether renders the operation safer for nervous patients and for those with heart involvement. It is true that the ether is discontinued immediately upon removal of the goitre; nevertheless, the reporter feels that Lahey's method speaks for itself and prefers to get along without ether wherever possible.

Another matter of interest was the correlation of the clinical symptoms with the pathologic findings. The subject of fetal adenomatous goitre, with its degeneration areas of softening and absorption of toxic material from these areas, is most interesting: the pathology of this condition has been quite fully worked up by Wilson, MacCarty and others of the Mayo Clinic, whose papers will be found in the annual publications from that source during the past few years. (P. G. S., Jr.)

CRUSH OF ARM: AMPUTATION UPPER THIRD UNDER NOVOCaine-BLOCKING OF
BRACHIAL PLEXUS BY KULENKAMPF'S METHOD

CASE V.—J. H., male, white, aged fifty-five, locomotive engineer, was admitted as an accident case to Polyclinic Hospital (Case Record No. 32,176) on October 19, 1917.

⁸ *Surg., Gynec. and Obst.*, vol. xxiv, No. 2, February, 1917, 229.

History of Present Condition.—The patient was run over by a train, receiving a crush of the right arm, a deep lacerated wound of the right buttock, and contusions of the head. He was admitted to the hospital in a state of shock, and was given morphine sulphate, gr. $\frac{1}{4}$, by hypodermic.

Physical examination reveals absence of the right forearm, exposure of the lower articular surface of the humerus, and a crush of the upper arm below its middle. Shortly after admission the patient had a severe chill, but this was overcome by elevating the lower extremities and applying hot-water bags.

Operation.—Preliminary intravenous infusion of 1000 Cc. of normal saline. Right brachial plexus blocked above clavicle with 20 Cc. of a one per cent. solution of novocaine. Crushed lower half of arm covered with towel. Brachial vessels exposed in upper third of arm and ligated. Amputation made through upper third by modified circular method, anteroposterior flaps. Medullary cavity of humerus iodized. One perforated rubber tube placed in angle of flaps and across bone-end. Muscles quilted with chromic gut. Deep fascia closed with continuous suture of chromic gut. Skin-edges apposed with interrupted sutures of silkworm-gut. One piece rubber dam introduced into subcutaneous space through stab-wound in posterior skin-flap. Stump irrigated with Dakin's fluid. Dry dressing. Buttock wound swabbed out with dichloramin-T and left open with rubber-tube drainage in depths.

Postoperative Course.—Owing to the presence of devitalized tissue in the stump, which was recognized at operation, the stump had to be reopened on the third day and was allowed to heal by granulations. The dichloramin-T absolutely prevented infection gaining a foothold in the deep buttock wound.

Here again the main point of interest lies in the mode of anaesthesia. The patient was so badly shocked from trauma and from loss of blood that the reporter did not deem it wise to anaesthetize with ether, so he thought himself of Kulenkampf's method. With the finger as a guide upon the subclavian artery just above the middle of the clavicle, the needle was pushed through the skin just to the outer side of the finger-guide, penetrating until the patient complained of paræsthesia (sharp, shooting electric pains radiating down limb). The needle was pushed in until it struck the first rib, which it reached at a depth of about one inch. The barrel of the syringe was now attached to the needle, and the injection was made as the needle was slowly withdrawn. Anaesthesia began immediately and was sufficiently advanced within five minutes to begin operation. The patient was conscious and chatted throughout the operation, at no time experiencing pain except when a narrow strip along the inner side of the arm was crossed by the knife—an area corresponding to the course and distribution of the intercosto-humeral nerve. Since this nerve receives a contribution from the second intercostal (sometimes the first), it was not, of course,

reached by the plexus injection; a local injection of a few drops of the novocaine soon solved the problem, however. Not only did this brachial plexus blocking serve to anaesthetize the limb, but it also intercepted shock-impulses (nociceptors) from the operative field, so that as the operation went along the patient's condition as regards shock gradually became better and better, whereas with ether anaesthesia and without nerve-blocking it is but logical to conclude that the opposite effect would have obtained. At the end of the operation the patient's pulse was 72.

In blocking the brachial plexus it is well to bear in mind that its component cords approach the clavicle not to spread out horizontally, as it is commonly conceived of doing, but arranged one behind another in the sagittal plane, thus forming a vertical partition having anterior and posterior edges and outer and inner sides, the practical application of this anatomic fact being that the needle when beneath the skin does not have to play from side to side to get all the cords of the plexus, but solely from before backward. The method is easy and accurate and should be more widely used.

In sewing up the stump care was taken to approximate the edges of deep fascia with a separate suture, since this layer is normally interposed between the bone (and muscle) on the one hand and the skin on the other: failure to observe this precaution means adherence of the bone to the skin and painful stump. Another cause of painful stump is exuberant callus-formation over the end of the bone: this can be prevented by removing the periosteum a little way back from the bone-end. A third cause of painful stump is involvement of the nerves in cicatricial tissue, or the formation of bulbous neuromata on their end: this can be prevented by pulling the nerves out, holding them, splitting the nerve-end in halves, and removing one-half for a little distance back from the end and suturing the long half around to the short-cut portion, thus allowing the axis-cylinders of one-half of the nerve to heal in continuity with those of the other half and preventing exposure of any axis-cylinder processes.

The modified circular method of amputation is winning a place for itself in the present European war. Its strongest point of recommendation is that it preserves the maximum length of bone in the stump—a consideration of much moment when it comes to applying an artificial limb to the stump. (P. G. S., Jr.)

ASCENDING NEURITIS OF FOREARM

CASE VI.—J. M., male, white, aged twenty-nine years, on December 26, 1916, cut the tip of his fourth finger on a milk bottle. When first seen, December 30, 1916, he complained of a throbbing pain in his finger, which, however, did not appear to be infected; the small wound was closed and did not exhibit any signs of inflammation. His finger was placed on a splint and various local remedies used to relieve the pain, but without any success. January 6 the pain persisted in the finger, and then extended up the arm, was constant, aching in character, and had prevented the patient from sleeping. He showed the effect of loss of sleep, was pale and worn out. On January 7 he noted two small pea-sized nodules on the flexor surface of the forearm: these were very painful on touching. Several red streaks were also present at this time; the condition was diagnosed as lymphangitis and the arm placed on a splint and kept moist with a saturated solution of magnesium sulphate. The external evidence of infection subsided in two days, but the pain persisted.

On January 10 he was referred to the neurologic clinic for examination by Dr. W. B. Cadwalader, whose notes state that there was no motor weakness of the hand, but movements excited pain. The tip of the injured finger was hyperæsthetic. The pain was dull, aching, throbbing, and was always worse at night. The skin of the whole forearm and hand was very sensitive—in fact, hyperæsthetic. There was no anaesthesia, no motor paralysis. Wassermann reaction negative.

A diagnosis of ascending neuritis was made, and on January 19, under novocaine anaesthesia, the small nodules referred to were removed. They were attached to a small superficial nerve, and were very painful until the nerve was blocked, when they were removed along with a portion of the nerve. Following this procedure, the pain was greatly relieved and was limited to the region of the thumb.

February 2, 1917.—The patient said he developed a sudden sharp pain in the wrist; it extended down the thumb to the tip, and at the same time the thumb felt dead. Sensation was impaired, but the dull ache persisted and was limited to the thumb. Over the base of the thumb cutaneous sensation was greatly impaired or entirely lost, but there was very severe pain on pressure. There was no motor paralysis.

There has been much debate among neurologists concerning the theory of an ascending neuritis, and many deny that such a syndrome exists. A study of the literature on the subject will, however, show that a sufficient number of clinically and anatomically proved cases have been reported to justify the assumption that an ascending neuritis exists.

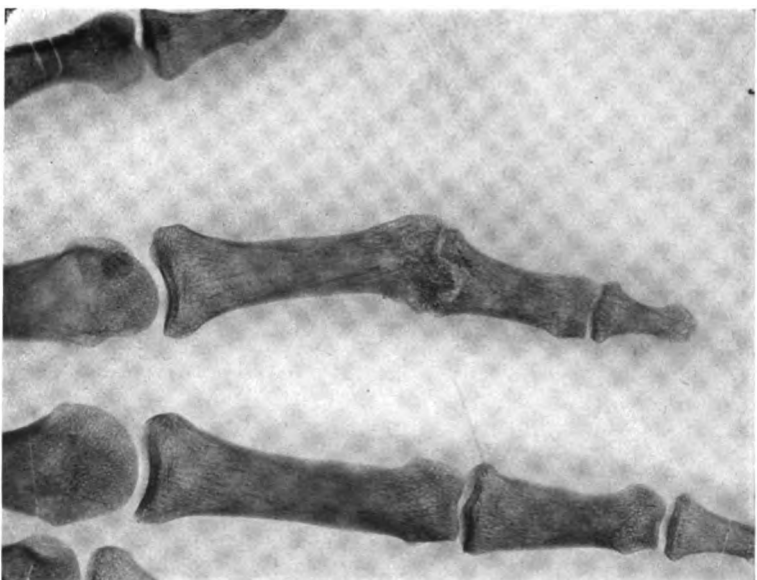
These cases have been collected by Cadwalader, who has observed five instances of ascending neuritis, including in his report the case we have described. He states that since the first case was demonstrated by Remak in 1861 the question has undergone alter-

nating periods of popularity and neglect. The cases of obscure character in which severe migratory pains were encountered were designated neuritis migrans, for no better reason, apparently, than because of diffuse or migratory pain, and without regard to the presence or absence of an open wound or a focus of infection.

The most concise clinical description found was that of Déjérine, who refers to muscular atrophy in so-called ascending neuritis, and states that the following symptomatology is sometimes observed clinically. Following a lesion of the tip of a finger, especially of the digital pulp, or of the palm of the hand—a lesion, often insignificant and hardly to be regarded as trauma, such, for instance, as a scratch of a pin or a needle or a slight cut with a bit of glass, more rarely a felon—there is sometimes seen more or less retarded cicatrization; at the same time the affected region becomes the seat of severe pain, which soon involves the entire finger and then extends to the other fingers, the palm of the hand, the forearm, etc. The digital pulp of the affected finger atrophies and the extremity assumes a conical shape, the skin is readily cyanosed and becomes cold, and gradually the skin of the entire hand becomes glossy and thin. There is severe pain in the entire arm, but chiefly in the region primarily injured. The intensity of the pain varies in different individuals; pressure on all the nerve-trunks of the upper extremity, the collaterals of the fingers up to the branches of the brachial plexus accessible in the subclavicular triangle, is exceedingly painful. This hyperexcitability of the nerve results in functional derangements, sometimes very marked, so that the patient is afraid to move the fingers or the hand for fear of exciting pain. The joints are likewise often painful and their movements are limited. The superficial sensibility is intact and at times increased. Finally, there is generally more or less atrophy of the muscles of the hand, the forearm and the arm; this manifests itself in painful phenomena, and occasionally may attain a most profound degree. The prognosis of this affection is sometimes very grave, and cases have been observed in which the total loss of use of the arm resulted.

In serious cases, in addition to the trophic disorders of the joints, there may be fibro-muscular retraction and elephantiasis of the paralyzed member; sometimes, however, the affection runs a mild course and recovery takes place within a few months.

Fig. 1



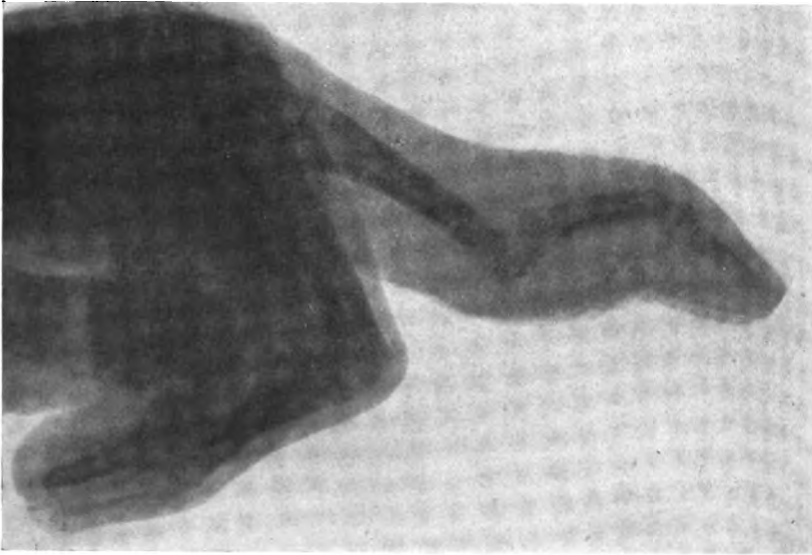
Ancient fracture-luxation before operation. Note hypertrophy of outer portion of head of proximal phalanx, inclining end of finger over toward ulnar side.

Fig. 2



Appearance of head of phalanx after wedge-shaped resection and arthroplasty. The articular condyles swung around from the anterior lip of the phalanx-head on a periosteal hinge to a position over the end of the bone are well shown, separated from the shaft by a clear line, which represents the plane of section after removal of the wedge; thus was the arthroplasty performed. The ulnar deviation of the finger-end was easily corrected by a lateral splint, which was applied when the condition was detected by the skingram.

FIG. 3



Ancient fracture-luxation before operation (lateral view). The fracture involved the head of the phalanx, which became greatly enlarged and deformed from excessive callus-formation. The patient could not flex or extend the finger.

FIG. 4



Ancient fracture-luxation after operation (lateral view). The joint is now supplied with two cartilage-coated ends. Finger dressed in semi-flexion to overcome the tendency to dorsal displacement. After healing had progressed to the point of stabilizing the joint, massage and passive motion were begun.

Of all the manifestations observed, pain was the first, most severe, and most constant symptom. The diffusion of pain above and far beyond the site of injury should suggest the ascending nature of the irritative lesion. In the differential diagnosis erythromelalgia and such conditions as thrombo-angitis obliterans must be considered.

The prognosis, as a rule, is rather unfavorable, because the usual methods of treatment do not relieve pain, which is the most important indication in treatment. The pain gradually subsides after a more or less indefinite time, regardless of the various measures used for its relief. But in the meantime the patient's general health becomes impaired and the constant suffering gives rise to a variety of neurotic, hysteric, and psychasthenic phenomena. No uniformly successful method for the relief of pain is known, although resection of the peripheral cutaneous nerve branches was beneficial in two of the cases observed by Cadwalader. (J. S.)

ANKYLOSIS OF FINGER DUE TO ANCIENT FRACTURE-LUXATION: RESECTION;
ARTHROPLASTY

CASE VII.—J. O'B., male, white, aged eighteen, single, clerk, was admitted to Polyclinic Hospital (Case Record No. 32,048) on September 29, 1917.

History of Present Condition.—On November 15, 1916—nearly one year ago—the patient caught his left index-finger in a machine: the skin was torn and the ends of the bone forming the proximal interphalangeal joint protruded through the wound. The bones were replaced and the wound was sutured: the patient stated that no infection followed. The joint was stiff and the patient sought operative relief to qualify for enlistment in navy.

Physical examination revealed partial luxation at this joint—the base of the middle phalanx was displaced dorsally for one-half the articular surface. The head of the proximal phalanx was hypertrophied, and on its outer side there was a boss projecting beneath the skin. Motion at this joint was limited to a very small range. The right index-finger was traumatically amputated through the middle of the middle phalanx.

Skilagrams showed hypertrophy of the head of the proximal phalanx (Fig. 1), with partial luxation of the base of the middle phalanx upon the head of the proximal (Fig. 3): the joint surfaces appeared roughened.

Operation.—Limb exsanguinated to above elbow: Esmarch tourniquet applied. Digital nerves blocked with two per cent. novocaine. Horseshoe skin-flaps—base above—raised from dorsum, exposing extensor tendon: latter divided and retracted. Joint opened by dividing lateral portions of capsule. Flexor tendons found intact. Joint surfaces had been united by fibrous tissue. A projecting ledge of bone was found at anterior portion of base of middle phalanx: this was removed with the saw. A rectangular wedge of bone was removed with a fine metacarpal saw from the head of the proximal phalanx in such

manner that the condyles on the head which faced anteriorly could be swung around on an anterior periosteal hinge until they faced directly distally, articulating with base of middle phalanx (Fig. 4). This articular portion of the head was secured by two chromic-gut periosteal sutures, which brought freshened bone surfaces in apposition. These articular surfaces were cartilage-coated and should not reunite. The capsule was closed laterally, and posteriorly it was closed by suturing the ends of the extensor tendon with chromic gut. Skin-flap restored and sutured with interrupted sutures of silkworm-gut. Skin swabbed with iodine. Alcohol gauze dressing. Dorsal splint applied, retaining finger in semi-flexed position, owing to tendency to luxate backward.

There is an immense untilled field for restorative surgery of digits crippled by trauma or the sequelæ of infection. Every one sees people carrying around mallet-fingers, fingers that can not be flexed and are in the road, fingers that can not be extended and are functionally useless, and fingers (and toes) that are distorted in other ways. For some unknown reason these people have been educated to take their wrecked fingers as a matter of course; seldom is any attempt made to restore the digit to usefulness by modern surgical means. Such a simple little operation as that described in our text case meant a great deal to this patient, the more because the index-finger of the opposite (right) hand had been traumatically amputated through the middle of the middle phalanx.

Of even greater importance are measures directed toward the conservation of fingers at the time of the primary operation for trauma or infection. Illustrative of the first class (*i.e.*, trauma) are the skiagrams appended (Figs. 5 and 6), which were taken shortly after operation: in both cases through each step of the operation the dominating thought was so to fashion each digit as to restore to it the maximum function attainable. As to the primary operation for infection, uniformly good results will not be attained until fingers and palms are opened in a more intelligent manner than heretofore. The work of Kanavel and others on palmar infections should be read and studied by everybody. Only recently the reporter had a case of flexion contracture of the finger due to violation of the surgical principle of never to make an incision across a joint flexure. As always occurs when this rule is violated, the scar contracted and contracted until the finger was drawn into the palm, and to correct the deformity it was necessary to excise the scar and transplant a pedicled skin and fat flap from the abdomen.

FIG. 5



Cases illustrating the conservative surgery of the hand and fingers. I. This patient caught his hand in a circular saw. The blade of the saw divided the interphalangeal joint of the thumb, the metacarpophalangeal joint of the index, the head of the metacarpal of the middle finger, and emerged through the neck of the metacarpal of the ring finger. The deepest cut involved the index, the flexor tendon being partially severed, and a portion of the articular surface of the metacarpal head being found on the floor by the patient. The hand was, in fact, literally cut in two from the dorsum to the palm. At operation four planes of suturing were necessary: (1) the joint-capsules; (2) the extensor tendons; (3) the deep fascia (to prevent sutured tendons adhering to skin); and (4) the fat and skin. This skiagram shows the condition after operation. Massage and passive motion were begun on the seventh day, after the trauma reaction had subsided and the stitches had been removed. Now, two months after operation, the patient's hand is rapidly limbering up and recovering its finer movements.

FIG. 6



Cases illustrating the conservative surgery of the hand and fingers. II. This patient caught his hand in a machine, losing as much of the fingers as the skiagram reveals. This picture, taken after operation, gives a good idea of the essentials in the con- or pre-servative surgery of the fingers. The thumb was not involved. The sharp edge of the stump of the index was snipped off with the bone-forceps, and the flexor and extensor tendon-ends were sutured together over the bone-stump and their sheaths were closed: note in the skiagram the dim outline of the copious cushion-flap fashioned for the index, affording the thumb an extensive surface for the valuable function of apposition. The head of the middle metacarpal was removed to allow eventual approximation of its neighbors. The phalanx-stump of the ring finger was preserved and the tendon-ends were sutured over it to give more strength to the little finger. The end of the little finger was restored for apposition with the thumb.

This patient was exhibited before the October, 1917, meeting of the Philadelphia Academy of Surgery. (P. G. S., Jr.)

RUPTURE OF LIVER OPPOSITE HEPATOCOLIC LIGAMENT: SUTURE; RECOVERY

CASE VIII.—A. R., male, colored, aged forty-four, was admitted to Polyclinic Hospital (Case Record No. 31,351) on May 23, 1917.

History of Accident.—Ten days previous to admission the patient was squeezed between two cars. He worked up to the time of admission, when he had severe abdominal pain, followed by vomiting and fever.

Physical examination revealed rigidity and tenderness of abdomen, most marked in right upper quadrant. Leucocytosis was present.

Operation.—After making a celiotomy incision, a laceration was found on the under surface of the liver, involving the hepatocolic ligament. An attempt was made to stitch the rent, but the sutures would not hold, so that a cigarette drain was placed down to the site of rupture. The blood-clots were sponged away. The intestines were found injected and beginning peritonitis was present. Drainage of the pelvic cavity was effected through a suprapubic stab-wound. The right rectus incision was closed. The patient made an uneventful recovery.

This case represents an unusual result of trauma. The tear in the liver was found in its visceral surface one inch above the gall-bladder, and was apparently due to a tear of the ligament between the liver and the hepatic flexure of the colon. That the patient was able to work for ten days following the accident without much discomfort was probably due to rapid sealing off of the site of rupture by the omenta: the later development of diffusing peritonitis was doubtless due to giving way of these walling-off structures during a sudden strain while at work. This injury can aptly be compared with that of tear-fracture of a bone. (J. S.)

PSEUDOMYXOMA OF PERITONEUM COMPLICATING INTESTINAL OBSTRUCTION:

CELIOTOMY; RECOVERY

CASE IX.—E. G., female, white, aged fifty-four, was admitted to Polyclinic Hospital (Case Record No. 30,659) on February 21, 1917.

Previous History.—The patient was operated upon five years previously for gall-stones.

Present Condition.—The patient was well until one week before admission, when she developed symptoms of intestinal obstruction—constipation followed by vomiting. Her bowels had not moved during that time.

Physical examination revealed a mass in the umbilical region resembling volvulus.

Operation.—After making a celiotomy incision through the right rectus muscle the small bowel was found adherent to the old gall-bladder operative field in such manner as to obstruct the hepatic flexure of the colon, producing marked

dilation of the cæcum, which was the size of a child's head. There was encountered in the right kidney pouch a jelly-like mass about the size of a billiard ball, which, on examination, was found to consist of pseudomyxomatous material. Gelatinous material of the same type was found scattered freely throughout the peritoneal cavity. (See frontispiece.)

Postoperative Course.—The wound healed *per primam* throughout, and the patient was discharged on March 15, 1917, cured.

The source of this pseudomyxomatous material was probably a pseudomucinous ovarian cyst which ruptured, causing extravasation of the contents into the peritoneum. The gelatinous material is spread everywhere over the wall and becomes partly organized by the upgrowth of granulation tissue. According to MacCallum, some writers have thought that epithelial cells are implanted and continue the production of the pseudomucin, but the evidence for this is insufficient. The result is that the whole peritoneal cavity is lined with a thick, translucent layer, often spoken of as *pseudomyxoma peritonei*. A similar process may take place in connection with certain epithelial tumor growths of the appendix vermiformis. (J. S.)

GANGRENE OF BOWEL FROM STRANGULATED INGUINAL HERNIA: ENTEROSTOMY FOLLOWED BY RESECTION; RECOVERY

CASE X.—L. C., male, negro, aged twenty, single, laborer, was admitted to Polyclinic Hospital on September 21, 1917.

Chief Complaints.—Lump in right groin and pains in abdomen.

Previous History.—The patient stated that since birth he had had a lump in the right groin: in recent years he had been compelled to wear a truss to keep the lump back. Some years ago this lump "came down" and caused much pain, but when the patient lay down in bed the lump went back and the pain disappeared. Two months ago he discarded his truss.

Present History.—Two days previous to admission, while the patient was doing laboring work, he coughed and the lump suddenly descended into the scrotum: he was unable to get it back. The lump became painful and the patient could not succeed in having a bowel movement. He had at no time had stercoraceous vomiting. The cough was the result of a recent cold he had contracted.

Physical examination revealed in the right groin a complete scrotal indirect inguinal hernia which was irreducible, tense, and evidently a strangulated enterocele. Powerful vermicular peristaltic waves were visible through the abdominal wall. The patient was nauseated, but did not vomit. His temperature, pulse and respiration, and general condition favored immediate operation. Rectal examination negative.

Operation No. 1 (September 22, 1917).—Ether. Usual inguinal herniorrhaphy incision made, exposing hernial sac. Unopened hernial sac drawn out of scrotum and opened: cloudly serous fluid escaped. There was now revealed a deeply congested loop of small bowel which was adherent by fresh exudate to

interior of sac: separation of the sac from the loop was easily accomplished, and the stricture at the neck of the sac was quickly divided. The loop of bowel was wrapped in cloths wrung out in hot water; after five or ten minutes' waiting the dusky color gradually changed to a dark pink, except at the lowest point of the convex border of the loop, where the peritoneum was very oedematous. The constriction rings at the neck of the loop were not only small, but very much thinned out. Owing to the dubious state of the gut-wall at these three points of the loop it was decided not to return the loop within the abdomen, but to secure it in the wound and drain the proximal bowel just above the proximal constriction ring. The loop was drawn out two inches and sutured to the parietal peritoneum. A small opening was made in the free border of the proximal loop two inches above the proximal constriction ring: in this opening a No. 26 rubber catheter was passed six inches and retained by a catgut stitch and also by a purse-string suture of silk. The catheter immediately began to drain dark-brown, thick, liquid intestinal contents and gas. The outer angle of the skin incision was closed with two interrupted sutures of silkworm-gut. Over the loop of bowel were placed cloths wrung in hot water, and over these cloths a hot-water bag was applied. To the distal end of the catheter there was attached a glass tube, and to this another longer rubber tube, which conveyed the intestinal contents into a bottle at the side of the bed.

Postoperative Note.—Four days after operation it was observed that the doubtful area on the free border of the lowest point of the convexity of the loop had undergone gangrene and become perforated longitudinally for a distance of one and one-half inches. The constriction rings had recovered themselves, being much larger than at the time of operation. The loop of bowel was of good color, but its peritoneal covering was roughened by exposure and irritation: fresh plastic lymph-exudate glued the loop to the edges of the incision made in the parietal peritoneum. The patient's condition being satisfactory, it was decided to resect the affected portion of bowel and perform end-to-end anastomosis.

Operation No. 2 (September 27, 1917).—Ether. Loop of bowel released by separating from it the edge of the incision made in the parietal peritoneum and covered with cloths wrung out in hot lysol, the whole mass being then covered with a towel. The loop was now pulled away from the abdominal wall until good bowel was exposed, there being withdrawn four inches of the proximal loop and the same length of the distal loop. At this point on each segment a small Scudder intestinal clamp was applied: on the loop side of each of these clamps and one and one-fourth inches distant from them a crushing clamp was applied. With the electric cautery the loop was severed between the Scudder and the crushing clamp, but close to the latter; and after ligating the mesentery close to the bowel with interlocking sutures of silk, the loop was cut away from the mesentery and removed. The ends of the bowel were disinfected with lysol. The rent in the mesentery was closed with silk. The ends were now approximated and end-to-end anastomosis was made. Posterior seromuscular silk line placed. Using a fine chromic-gut suture, the dead space of the mesentery was next obliterated by Lee's stitch: the ends of this thread were left long; one end was used to approximate the near cut edges of the bowel by a buttonhole suture, while the other end of the suture approximated the far cut edges by a "mucosa-inverting" or continuous Connell stitch: these ends were tied at the free border of the bowel, the knot slipping within the lumen. The clamps were now removed and the posterior silk suture was now completed anteriorly, using Cush-

ing's right-angled stitch. Intestine replaced in abdomen; split tube inserted into pelvis; one piece of coffer-dam gauze placed in wound to prevent evisceration of bowel.

Postoperative Course.—At no time after operation did the patient's temperature exceed 98 $\frac{1}{4}$ ° F., nor did the pulse rise above 60. On the third day after operation an enema was given, and there was a very copious result. The patient was kept on his back for one month to get sound wound-healing of the hernial site, reconstruction of the canal by suture being rendered impossible by infection of the parietal tissues by the enterostomy loop of bowel.

To estimate with any degree of accuracy the viability of a loop of bowel that has been strangulated for some time is often a very difficult matter. In our text case at the first operation the doubtful area was at the lowest point of the convex border of the loop, and the only sign that raised the doubt as to the viability of the bowel was the oedema of the peritoneum at this site. That this doubt was justified after-events proved, for by the end of the fourth day after operation this oedematous area had given way completely from gangrene, making a natural slit one inch long, which added a second enterostomy opening to the one intentionally made at the first operation. Had the bowel been returned at the first operation and the hernial wound closed without drainage the results would have been most disastrous. Primary resection was not performed owing to the patient's precarious condition.

But a doubtful loop of bowel may be returned within the abdomen (in a case of strangulated inguinal or femoral hernia), provided the wound is left wide open for drainage; such a coil of bowel seldom travels far from the hernial ring, and if the gut at a later period gives way, it will do so gradually; and, as adhesions are rapidly formed, the intestinal contents will escape along the course of the open sac. It seems, however, that when primary resection can not be performed the preferable method is the one employed above; namely, open the sac, divide the stricture, and leave the gut *in situ*, first securing it by means of two or three silk stitches passed through the serous and muscular coats, and then fastened to the skin.

In every case of advanced acute intestinal obstruction it is of prime importance to establish immediate drainage of the small bowel, whose contents—especially those of the jejunum—are highly toxic: it was for this reason that the proximal loop was drained by a tube. (P. G. S., Jr.)

ACUTE PYOGENIC OSTEOMYELITIS OF ILIUM WITH ILIAC FOSSA ABSCESS: TREPHINE OF ILIUM; RECOVERY

B. M., male, white, aged seven, schoolboy, referred by Dr. Bernhard Segal, was admitted to Polyclinic Hospital (Case Record No. 31,923) on September 2, 1917.

Present Illness.—The patient was taken suddenly ill one week previous to admission. Owing to the child's age the subjective symptoms were not readily elicited, but the child complained of pain in the right groin. The family physician stated that the child vomited once only—on the fifth day—and that his bowels had been moving up to the day before admission.

Physical examination revealed: Temperature, 104° F.; pulse, 140; respiration, 36. The lungs were negative. The abdomen was neither rigid nor tender, and no masses could be felt within the abdomen. Rectal palpation revealed no induration in pelvis. It was observed that the child "favored" the right hip, and that the right thigh was slightly flexed on the abdomen. There was fullness just below Poupart's ligament, in the outer portion of Scarpa's triangle: over this area there was subcutaneous oedema which pitted on pressure, and pressure upon which caused tenderness. The thigh could be moved at the hip-joint without much resistance.

Interpretation.—There was a phlegmon on the right side between the neck of the femur and the dorsum of the ilium.

Operation No. 1 (September 2, 1917).—Ether. Incision begun just below and to inner side of anterior-superior spine of ilium and passed downward and slightly inward for four inches. On dividing skin oedema of superficial fascia was noted. Superficial vessels clamped and ligated. Fascia lata divided at outer border of sartorius; sartorius and rectus femoris retracted inward; tensor fascia latae and gluteus medius retracted outward. Working down in this interval, a walled-off abscess the size of a lemon was opened: this abscess lay between the top of the great trochanter and dorsum of the ilium. No distention of capsule of hip-joint was found, nor could the presence of diseased bone be determined by palpation. The capsule of the hip-joint was not incised. The pus was evacuated and drainage inserted, consisting of one rubber tube, one piece of gauze, and one piece of rubber dam. Buck's traction applied.

Postoperative Course (September 10, 1917).—A subcutaneous abscess over the right scapula was opened and drained. Bacteriologic examination of pus from hip and from this abscess showed the same type of organism—*S. pyogenes aureus*. Skiagram of right hip showed no evidence of bone disease.

(September 14).—Temperature rose to 103 2/5° F. at five o'clock yesterday afternoon: this morning's temperature was 101° F. In searching for the cause of this fever there were found over the left external malleolus swelling, redness, and tenderness.

Interpretation.—Metastatic epiphysitis of lower epiphysis of left fibula. This, together with the superficial scapular abscess previously opened, established the presence of staphylococcic septicæmia, or bacteriæmia. An autogenous vaccine was made up.

Operation No. 2 (September 14, 1917).—Gas. Two-inch vertical incision made over left external malleolus: periosteum incised—pus escaped. Cancellous tissue of metaphysis as well as of epiphysis contained pus: this was chiselled away until healthy bone was reached. Cavity flooded with iodine and then packed with iodoform gauze.

Postoperative Course.—Bacteriologic examination of pus from malleolar abscess showed same type of organism.

(September 15).—Patient seems greatly improved. Buck's traction apparatus removed.

(October 3).—Temperature is of septic type—high at eight o'clock in the evening and low at four in the morning. Wounds in gluteal region inspected: all found filled up with healthy granulation tissue. There are tenderness and fullness in right iliac fossa. Skiagram revealed bony proliferation at and around site of first abscess and around wing of ilium.

Interpretation.—In the presence of the healthy condition of the tissues at the site of the first operation and in the absence of evidence of a suppurative focus elsewhere in the body; considering the fullness and tenderness in the right iliac fossa and the skiagraphic evidence of disease of the right ilium, it was presumed that an abscess had formed on the upper surface of the ilium, just as the first abscess formed on its lower surface, and that this abscess, being confined, was giving rise to the septic fever. It seemed advisable to explore the right iliac fossa through a trephine opening in the iliac wing.

Operation No. 3 (October 4, 1917).—Gas. Long incision made along iliac crest from anterior-superior to posterior-superior iliac spine. Flap, consisting of skin, the two fascias, and the gluteus maximus and medius muscles, was elevated as far as acetabulum and turned downward. With the Hudson bur the iliac wing was trephined at its centre, evacuating about half an ounce of brownish pus (Fig. 7). The under surface of the iliacus muscle was exposed. The abscess cavity was irrigated with Dakin's solution and the wound was left wide open, being lightly packed. Culture taken from pus.

Postoperative Course (October 24).—The extensive wound is filling up rapidly with healthy granulations: one piece of sequestrum was removed to-day: it was about the size and shape of a signet ring. After the third operation the patient's temperature gradually came down to normal, but the pulse continued somewhat rapid. Autogenous vaccine is administered once a week. Patient kept in open air and fed six eggs and one quart of milk daily: he gains weight at the rate of one pound a week.

(November 6).—No further febrile disturbances. Pulse about 100. Right lower extremity is being massaged and moved passively; no impairment of hip-joint. Large wound over right hip nearly closed. Patient rapidly recovering lost flesh and will be discharged from hospital within a few days.

As in all cases of acute osteomyelitis with bacteræmia in childhood, this case proved a constant source of anxiety to the reporter. The boy was the only child, and the importunities of the parents and relatives can well be imagined. And then, owing to the presence of bacteræmia, the questions always loomed up: What was going to happen next? In what bone will the next embolic focus develop?

It is known that acute osteomyelitis does not manifest itself in skiagrams at an early stage, as comparison of the skiagrams here-

FIG. 7



Trephine of ilium to evacuate iliac-fossa abscess and afford drainage to osteomyelitis of ilium. The opening was made with the Hudson bur, and pus immediately escaped from the iliac fossa. Note extensive size of flap: this wound was left open and packed.

with presented will show. There was some doubt, therefore, after the first abscess was evacuated, as to just which component of the hip-joint was involved, if any. Was it a mistake not to open the hip-joint early? It was thought not, because if the first abscess had escaped from the joint-capsule there was already a natural opening present. And then the clinical signs did not exactly correspond with those presented by hip-joint involvement, such as acute epiphysitis of the head of the femur. When the skiagram finally revealed disease of the dorsum of the ilium, however, with no involvement of the hip-joint, many doubts and uncertainties were cleared away; and when the wing of the ilium was trephined and the iliac fossa abscess was evacuated, the active disease process was finally brought to an end, the septicæmia gradually cleared up, and the entire clinical picture changed from one of doubt and desperation to that of recovery.

In the *Annals of Surgery* (vol. xxi, p. 240) there is an abstract of a paper by von Bergmann on resection of the ilium for osteomyelitis, in which the author urges radical measures for this disease. The patient of the text was in no condition to withstand such an extensive operation, and there was no evidence of a sequestrum involving the entire wing of the ilium. It was thought that trephining would not only evacuate the iliac abscess, but also open the spaces in the cancellous bone of the dorsum of the ilium, just as in the mastoid operation. Both these anticipations were realized, the former when pus was encountered confined in the iliac fossa, the latter by the gradual subsidence of the fever and by the removal of the signet-ring sequestrum from the bony tissue of the dorsum of the ilium.

The entire picture presented by the disease formed a very interesting and instructive study in surgical pathology. (P. G. S., Jr.)

Medicine

A STUDY OF FIFTEEN CASES OF BRAIN TUMOR OF OBSCURE LOCALIZATION

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IN considering the subject of brain tumors and their localization one is immediately beset with many difficulties. In order to eliminate at once some of these, the author confines himself in this paper to the report of only those cases which have been traced from first diagnosis to demonstration of localization, either by operation or autopsy. To further restrict the number of cases there are here reported only those cases which present some unusual problem, either as to diagnosis of brain tumor *per se* or its localization.

Neurologic literature teems with papers on and reports of cases of brain tumor. Many of these publications are over-elaborate, theoretical and hypertechnical; often they are pessimistic, non-stimulating, and impractical. It may be that the writer will be scourged by his own lash, but, at any rate, his endeavor is to present a practical paper as succinctly as possible.

Many erroneous ideas of intracranial growths have found wide credence, possibly not among neurologists, but among physicians in general. One of these ideas is the belief that brain tumors cause increased blood-pressure, whereas, in fact, brain tumors have no real effect on blood-pressure. Frazier¹ has stated he was "unable to find anywhere that the blood-pressure was much higher (in brain tumors) than it might have been under normal conditions." Another erroneous idea is that it is dangerous to perform lumbar puncture in a case of brain tumor, especially if the growth is subtentorial. This the author does not believe, and he has never seen anything but temporary improvement follow lumbar puncture in a case of brain

¹ FRAZIER, CHAS.: "The Cerebrospinal Fluid and Its Relation to Brain Tumors," *New York Medical Journal*, June 27, 1914.

tumor, subtentorial or otherwise. Another error is to suppose that it is necessary, in making the diagnosis of brain tumor, to have the "cardinal symptoms" of increased intracranial pressure present; i.e., vomiting, vertigo, headache, and optic neuritis or choked disk. Weisenberg² has said that tumors in any part of the brain may exist without causing any of the so-called general pressure symptoms. The fact is that one or all of these symptoms may be absent until quite late in the progress of the growth, and even occasionally none of them ever become pronounced.

Radiography has been very little used in the diagnosis of brain tumor, but at times it is distinctly valuable. Another thing to be considered is that we usually think of a brain tumor as giving symptoms from only one hemisphere, whereas the growth may be multiple, may give by-hemispherical symptoms from pressure, or the same tumor may actually invade both hemispheres. Brain tumors are also more frequent than is generally assumed. In the author's private neurological practice they have occurred about 15 times per 1000 patients.

There is a general impression that operation offers practically no hope in the treatment of brain tumors except in occasional cysts. Every now and then, however, a case of other form of intracranial growth is operated upon with a permanently successful result. This is especially true of cerebellar tumors. Sinkler³ quotes Frazier, who collected reports of 116 cases of operation on the cerebellum for tumor, with recovery of 15 per cent., and Starr, who gave the result of 58 operations, with recovery of 16 per cent. This renders surgery directed to the removal of the growth, rather than limited to decompression, justifiable. If a man saw a hundred men drowning, and there was a chance of his saving one of them, he would be negligent indeed not to make the attempt, provided, of course, he was an expert swimmer and knew the principles of life saving. The success of brain surgery, so far as tumors are concerned, depends largely upon an early diagnosis, an accessible location, the nature

² WEISENBERG, T. H.: "Cerebellar Pontile Tumor Diagnosed for Six Years as Tic Douloureux," *Journal of the American Medical Association*, May 14, 1910, vol. 54, p. 1600.

³ SINKLER, WHARTON: "Three Cases of Cerebellar Tumor," *Ibid.*, September 26, 1908, vol. 51, p. 1057.

of the growth, and the skill of the surgeon. Bolder excision of pathologic brain tissue, the use of radium and even of the actual cautery, may offer more hope for the cases in the future. There is need of a closer study of the relation of blows on the head and brain tumors with the idea in mind that a prompt decompression might, by relief of pressure on lacerated brain tissue, prevent the possible origin of some intracranial new-growths.

It might be thought that the diagnosis and localization of brain tumors is a matter solely of a knowledge of the anatomy of the brain and of precision in interpreting organic symptoms. It is true that these are two essentials, but in addition one must use general medical knowledge, intuition, deduction, and imagination.

The above and other points are illustrated in the following case reports:

CASE I.—Frontal Lobe Tumor Involving Both Hemispheres, But Chiefly the Left, Invading the Pituitary Body.

White, single, female, forty-four years of age. Father died as a young man of heart trouble. Mother alive, but hemiplegic. One brother drank considerably as a young man; one sister well, but rather neurotic; another brother apparently normal. There has been considerable deafness in the family. The patient has been somewhat deaf, especially in the left ear, for years. The patient is said to have grown nine inches the summer she was sixteen years old. She was operated on in July, 1915, for appendicitis.

During August, 1915, she began to complain of headache and to become more or less nervous and irritable. During November, 1915, she developed occasional nausea and vomiting, and shortly afterward vertigo. She was first seen December 7, 1916. She had lapses of memory, a desire to bathe many times a day, inattentiveness, and would stand at the window, gazing out for long periods of time. She was thought by her physician to be simply nervous and hysterical.

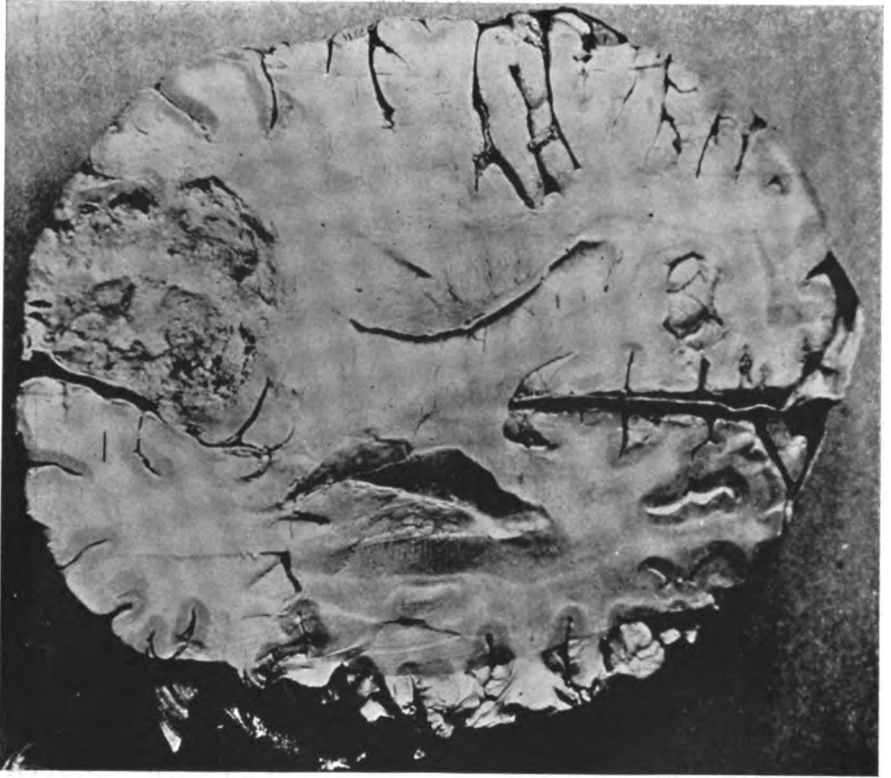
Her headaches were chiefly on the left side, radiating toward the vertex and down the neck. Her eye reflexes were normal. There was very slight exophthalmos, especially in the left eye. She had no clonus and no Babinski, but her knee-jerks were both exaggerated and the glossolabial fold on the right side was less pronounced. Her blood-pressure was 130 systolic. In a few weeks her headaches increased. She had occasionally nausea, vomiting, and vertigo, and

FIG. 1



Case I. Radiograph showing enlarged sella turcica in a neighborhood pituitary tumor of frontal lobe.

FIG. 2



Case I. Showing a large frontal tumor.

she developed an insatiate curiosity. The defect in memory continued. About the first of January, 1916, she developed a marked clonic spasm of the right arm and slightly of the right leg, and her gait became less steady. A lumbar puncture was performed and the fluid was found under slightly increased pressure, and gave a negative Wassermann, no cells, and a strong globulin reaction. Doctor Wright examined her eyes and reported the disks normal, but the field slightly concentrically restricted, both for form and color. The patient was of rather large growth, had prominent cheek-bones, and her complexion was somewhat pasty. Doctor Talley made an X-ray of the pituitary fossa, which he found enlarged, especially in the transverse diameter. The posterior clinoid processes were very hazy in appearance, giving the impression of partial destruction, and were somewhat distorted laterally. The floor of the fossa was very low and showed some bone destruction (Fig. 1).

On seeing her again Doctor Wright observed a slight nystagmus, but we were never able to elicit this afterward. Her urinalysis was normal.

Another examination on January 10, 1916, showed her neurologic and general symptoms the same as before, except that her station was slightly unsteady and the clonic tremor of the right arm was more marked. On January 20 she was sent to Doctor Frazier in Philadelphia with the diagnosis of a deep-seated tumor pressing on the left side of the pituitary fossa. A consulting neurologist there thought the whole condition was functional, and a reëxamination of her eyes failed to reveal contracted fields.

The patient was sent back to Richmond for further observation and treatment along functional lines. She was reëxamined and no change in the diagnosis of tumor was made, for conditions were the same as when she left, except that she was more drowsy and possibly dragged her right foot more. On February 21 she had a typical convulsion. Her eyes were reëxamined, and it was found that she had three diopters of choked disk in the right eye and one in the left.

The patient went again to Doctor Frazier for operation. A right subtemporal decompression was performed March 9, 1916. The brain was found under greatly increased pressure. The patient died in a few weeks. The post-mortem examination showed an endothelioma involving almost the entire frontal region of the left hemisphere

and largely that of the right. It reached from the base of the brain almost to the cortex and was three inches in diameter. Microscopic examination showed many moderate-sized cells, somewhat spindle in shape, with deeply stained nuclei. Dense cellular processes extended into brain tissue (Fig. 2).

Remarks.—There are many features in the consideration of this case which are of interest. Both early and late in the study of the case one good physician and then another made the diagnosis of hysteria. This is, of course, not an infrequent mistake in careless examinations, but here the mental attitude, the clonic spasm of the right arm, the concentric restriction of both the form and color fields, and the fact that the signs of increased intracranial pressure, except headache, were not very marked were what confused them. On the other hand, the fact that the growth was in the frontal lobe explained the mental symptoms, while the clonic spasm is best accounted for by the lesion interrupting the fibres from the frontal lobe to the red nucleus (Purves Stewart). Mills⁴ made the observation that "lesions of the tonectic cortex (chiefly the mid-frontal cortex) or of the striatum will give aberrant tonectic manifestations." It should also be remembered that concentric restriction of the fields is more often organic than functional.

The diagnosis of a neighborhood pituitary growth was partly based upon the radiographic findings. We have a long-standing pituitary history in the remarkable statement, which must be taken as more or less correct because it was investigated as far as possible, that the patient grew nine inches the summer she was sixteen years of age. Purves Stewart⁵ observes that in most cases of hypophyseal tumor the general signs of increased intracranial pressure are slight, and also the signs of acromegaly are frequently absent.

Other points to be noted in this case are that the blood-pressure was normal, that the onset of vomiting, vertigo, and choked disks was late, that the lesion involved both hemispheres, and that the patient showed marked curiosity. The writer has observed curiosity as a symptom in several frontal lobe lesions. Another interesting

⁴ MILLS, CHAS. K.: "Some Clinical Studies of the Problems of Cerebral Tone," *Ibid.*, November 18, 1916, vol. 67, p. 1485.

⁵ STEWART, PURVES: "Four Cases of Tumor in the Region of the Hypophysis Cerebri," *Review of Neurology and Psychiatry*, April, 1909, p. 237.

point is that the signs of increased intracranial pressure are usually slight in infiltrating growths.

CASE II.—Tumor of the Hypophysis in a Case of Acromegaly, with Marked Mental Depression and a Familial Pituitary History.

Married, white, female, age thirty-nine. Had four children, one of whom is nervous. Her father became insane and died at the age of sixty in an asylum. The patient's mother was noted for her very large bones, features, hands, and feet. One of the patient's children, a girl of eight years, began to menstruate at two years of age. This child weighed $84\frac{1}{2}$ pounds and was four feet nine inches tall. She has had full growth of hair on pubes since six years of age. Her breasts were the size of a girl of eighteen years of age, and she was very precocious at school.

The patient had been a healthy woman except for a spell of mental depression in 1895.

She was referred to me because she was again depressed, had lost interest, had ideas of unworthiness, and had threatened suicide. Her menstruation was regular, she was constipated, had headache, a poor appetite, and insomnia. These symptoms had lasted several months, but had been worse for a month.

Examination revealed the fact that the patient's features were large and coarse. Her shoes had increased from size $4\frac{1}{2}$ to $7\frac{1}{2}$ and her gloves from $6\frac{1}{4}$ to 8. This change was insidious, but was thought to have occurred in the five years previous to examination. Her fundi were normal. There was no diplopia, nystagmus, hemianopia or exophthalmos. Her pulse was 100. Her thyroid gland was enlarged, hardened, and symmetrical. She had no tremors, her reflexes were unaffected, and her urine was negative. A radiograph by Doctor Talley showed the walls of the skull thick, all accessory sinuses very large, and the pituitary fossa much enlarged, both in depth and anteroposterior measurements, with the posterior clinoid processes elongated and thin.

The diagnosis of a tumor of the pituitary body was made and the case was sent to Doctor Cushing for operation. A pituitary tumor was found and removed, and the patient was making a good recovery and her depression seemed less, but she jumped one morning, three weeks after the operation, from the third-story window of her home and died almost instantly.

Remarks.—Attention should be called to the fact that pituitary disturbance occurred in three generations in the females of this family—the mother, the patient, and her daughter. Another unusual thing is the occurrence of marked mental depression which led eventually to suicide. We note that the radiographic evidence was indicative of tumor, although examinations of the vision fields and fundi were normal and vomiting and vertigo were not present. Another feature of interest is that the thyroid was large and hard, without signs of exophthalmos, nor were there signs of hyperthyroid secretion.

CASE III.—Cystic Tumor of the Right Subcortical Motor Region, with Symptoms Resembling Cerebral Thrombus.

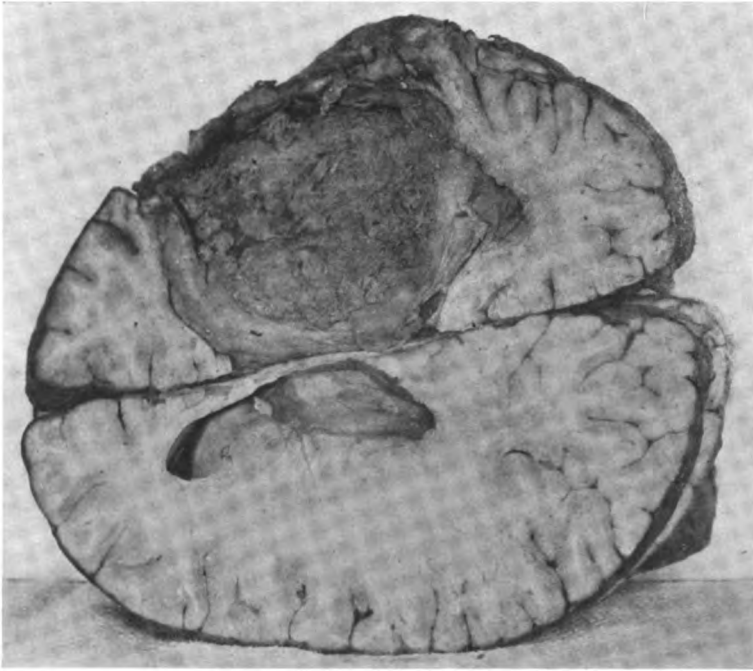
Patient first seen December 28, 1814, was white, age four years. Family history negative, except father had convulsions as a child. The past history was negative, except he always had a slight speech defect and two years previous fell from a second-story window. He was unconscious thirty minutes, but did not seem to have struck his head. Six weeks before he came under observation he began to lose use of his left arm and leg and also of the left side of his face. His leg became worse, but the condition of the arm and face remained stationary.

He complained of poor appetite. He had no headache, nausea, or vomiting. His cranial nerves were negative, except for weakness of the left seventh. Besides the partial hemiplegia, there were athetoid movements and moderate atrophy of the left arm. His reflexes were normal, except in the left leg the knee-jerk was exaggerated. Ankle clonus and Babinski were present on the left. There was no astereognosis. The Wassermann reaction was negative, urine examination and examination of his cerebrospinal fluid were negative. His sensation was normal.

Because no symptoms of increased intracranial pressure were present, and because of the gradual onset of the motor involvement, a diagnosis of cerebral thrombus affecting the right internal capsule was made.

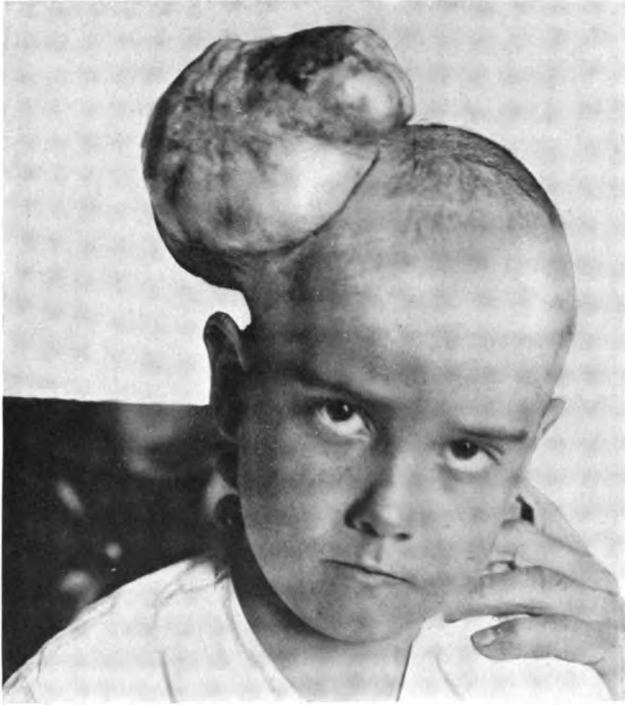
The boy went home and was brought back in three and a half months complaining of severe headache on the right side of the head and forehead. He also suffered greatly with nausea and vomiting. Examination showed his fundi still negative. There was no nystagmus, diplopia, or exophthalmos. A lumbar puncture showed the fluid

FIG. 3



Case III. Showing an enormous cystic tumor of the right subcortical motor area obliterating the right lateral ventricle.

FIG. 4



Case III. Showing large cerebral hernia. This child lived over a year with this hernia in comparative comfort and activity.

was under great pressure. Examination of the fluid, including a Wassermann reaction, was negative. Mental symptoms were absent. The diagnosis of a tumor in the right motor area was now made and operation advised.

On April 19, 1915, Dr. Stuart McGuire operated over the right motor area and found the brain under greatly increased tension, and it was thought that there was evidence of a deep-seated subcortical tumor. No evidence of a fracture was found. The wound was closed, the patient returned home, and a large cerebral hernia developed. His headache and vomiting ceased. In December, 1915, he was again operated on and a great cyst holding a half-pint of semi-gelatinous fluid was evacuated. After this his hemiplegia and speech improved. His hernia cerebri, however, increased in size until its walls became so thin that puncturing the cyst was again advised a year later. Twenty ounces of the same sort of fluid was removed, but the boy died in two days.

An autopsy was obtained and the growth was found to be an infiltrating glioma, more or less round, about $2\frac{1}{2}$ inches in diameter. It was cystic, could not have been removed by operation, and pressed on the right lateral ventricle until it practically obliterated it (Fig. 3).

Remarks.—In this case a child with the history of an injury two years previous developed a left hemiplegia of rather gradual onset six weeks before he came under observation. The relation of the injury, though remote, was probably definite. At first he had no signs of increased intracranial pressure, and the diagnosis of thrombus was made. In three and a half months localized headache, nausea, and vomiting had appeared, and the diagnosis was changed to tumor. Eye symptoms never appeared, probably at first because of the decompression and later because of the hernia's relieving the intracranial pressure. The size of the hernia (see Fig. 4) and the amount of the fluid in the cyst were the other remarkable features of this case. At the first operation the lesion was subcortical and the gelatinous fluid was obtained about an inch below the cortical surface. At the second operation the fluid was just under the skin over the cerebral hernia. The operations gave the boy symptomatic relief for over a year.

CASE IV.—*A Left Temporal Tumor and a Hemorrhage in the*

Opposite Hemisphere in the Subcortical Motor Area in a Patient with a Positive Blood Wassermann.

Seen June, 1915, in consultation with Dr. Frederic Hanes and reported by him.⁶ Male, single, age thirty-seven. Past history negative, except for lues 12 years previously. Present history: three months before he came under observation he had loss of power involving the whole left side, and unconsciousness which lasted ten days. For a year, his sister stated, he had headache and occasional nausea and vomiting. Mentally he had become lethargic.

Examination revealed a well-nourished man, hemiplegic on the left side. His sensation was normal, his reflexes were increased on the left, and Babinski and clonus were present on this side. His speech was hesitating, but not aphasic or parietic. His pupils were equal and reacted to light and accommodation. He had occasional diplopia, but no ptosis. He had double choked disk, greater on the right. The left side of his face was weak and his tongue protruded to the right.

Mentally he was stuporous. When aroused he laughed easily, but his memory and attention were poor.

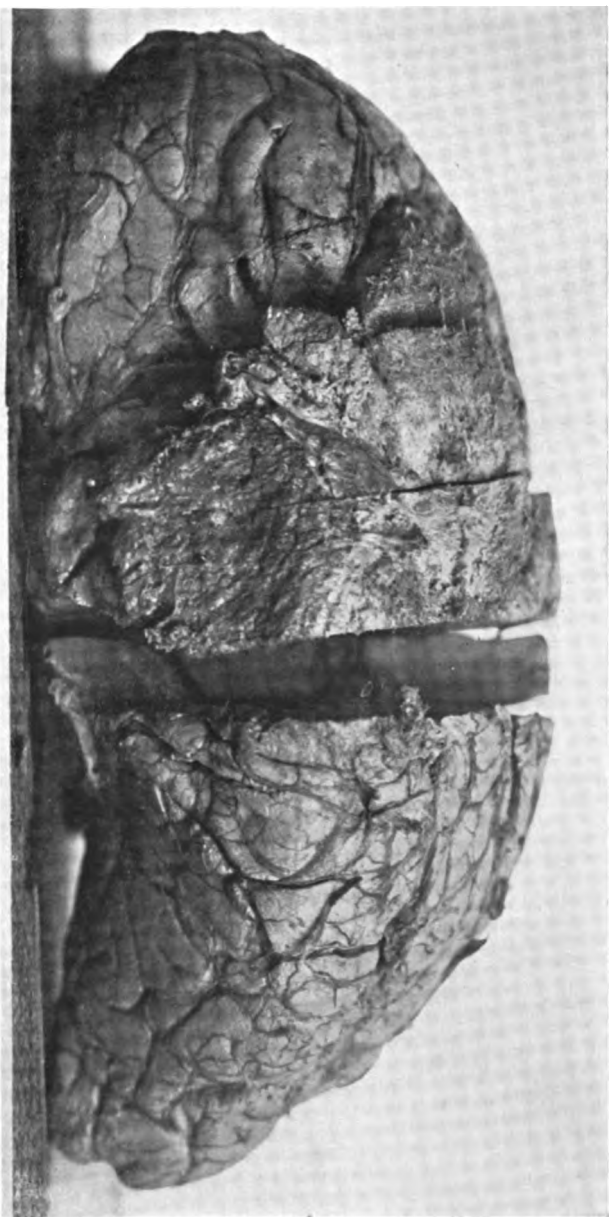
Examination of his urine was negative. His blood-pressure was negative. A Wassermann on his blood was strongly positive. A lumbar puncture showed his fluid under increased pressure, nine cells, and a negative Wassermann.

He was given salvarsan, but did not improve. In a few days a right subtemporal decompression was performed and the brain explored. The brain was under greatly increased pressure, but no other evidence of tumor was found. He died the next day. Examination of the brain revealed an endothelioma, three inches long by one and one-half inches in its widest diameter, the shape of a Bartlett pear, in the *left* hemisphere, the same side on which was his paralysis, and a subcortical hemorrhage on the right interfering with the motor fibres.

Remarks.—Here was a case of *left-sided* hemiplegia in a man thirty-seven years of age with a positive syphilitic history and a positive blood Wassermann. He also had mental symptoms. He did not improve on salvarsan and other anti-syphilitic measures, so it was naturally supposed that he had a lesion, probably a gumma,

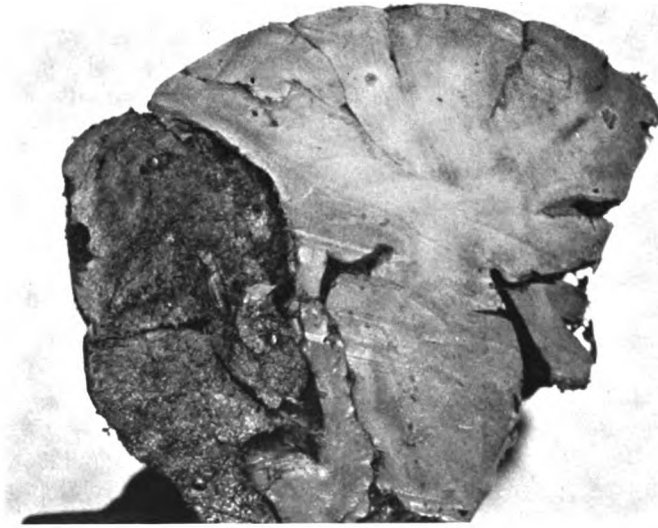
⁶ HANES, DR. FREDERIC M.: *Old Dominion Journal of Medicine and Surgery*, vol. 21, October, 1915.

FIG. 5



Case IV. Left temporal tumor with a section taken out.

FIG. 6



Case IV. View of a section of left temporal tumor, showing character of the growth.

FIG. 7



Case IV. Showing hemorrhagic area in the right hemisphere of a case in which there was a tumor in the left hemisphere.

involving the right motor fibres. Instead, autopsy showed a right subcortical hemorrhage under the motor area to account for his hemiplegia, and in the other hemisphere a tumor in the temporal region to account for his choked disk, speech disturbance, and increased fluid pressure. Interesting, also, is the fact that this tumor was an endothelioma and showed no evidence of syphilitic origin. We may assume, then, that this man had three conditions: lues, cerebral hemorrhage, and an endothelioma of the brain. Of course, it is probable that the hemorrhage was due to syphilitic vascular disturbance or lesion (Figs. 5, 6, and 7).

CASE V.—Tumor of the Pineal Body on the Left Side in an Adult, with Bilateral Headache and Bilateral Pain in the Face.

Married, male, age thirty-one, with two healthy children. He was first seen on October 21, 1915. His only complaint at that time was constipation and a bilateral pain which started at the back of the neck and ran over the head into the face and which had been diagnosed migraine. He had malaria when fifteen years of age, typhoid when twenty-two, and an attack of pleurisy when twenty-nine. His habits were good. He was constipated. There was no nausea or vomiting and rarely vertigo; his sleep was poor, his appetite good, and he never had convulsions or unconscious spells. At times he had polyuria. His nourishment was good and he was gaining in weight; his throat was slightly congested, his gums showed some pyorrhœa, and his tongue was coated. His nose had felt congested on one side.

His neurologic examination proved his station good, gait normal, knee-jerks normal, no clonus, no Babinski, no tremor, and sensation normal. His pupils were dilated, but reacted to light and accommodation; eye grounds normal, no nystagmus; no exophthalmos; no ptosis or diplopia. The mental examination was negative, except that he was rather verbose, argumentative, and forgetful. His blood-pressure was 118 systolic, 79 diastolic.

The patient was thought at that time to have a bilateral facial neuralgia, was put on salicylate, and was not seen again by me until January 20, 1916.

During the early part of October, 1915, he consulted a neurologist elsewhere, who thought his headaches were due to toxæmia, and reported that his optic disks were clear and there were no organic

neurologic signs. The patient then continued his occupation as a clerk in a railroad office until the first part of January, 1916, when he consulted a second neurologist elsewhere, who reported that he thought the patient had a nervous breakdown and that, "although there is some slight congestion of the optic nerves, the condition, I believe, shows no increase of intracranial pressure, and otherwise I determined no evidence of nervous trouble." He goes on to say that "his mental state is, however, odd, and this feature gives me some anxiety. He was confused, forgetful, and had a tendency to be argumentative and seemed a bit unreasonable. This phase of the condition suggested to me that he might be under the influence of some drugs. The only data I could get on that line were that he was taking aspirin quite freely."

When he returned to Richmond, January 20, 1916, there was still no nausea or vomiting, but slight vomiting. The station and gait were normal. The left pupil was slightly larger than the right, but reacted sluggishly to light. There were a few nystagmoid jerkings on looking in all directions. There was no ptosis. The left nasal fold seemed more marked than on the right, although movement of the face was well executed on both sides. The tongue protruded to the midline with no tremor.

The upper extremities showed movements full and equal in all directions, with good muscular tone, grip normal, finger-to-nose test normal, no diadokokinesia, reflexes equal on both sides. No tremors, abdominal and epigastric reflexes absent, cremasterics absent, and sensibilities intact. The lower extremities showed movements free in all directions, power equal to muscular development, and no atrophy or fibrillations. The deep reflexes on the left seemed slightly more exaggerated than the right. There was no Babinski and no clonus.

On January 22, 1916, the urine was examined and found negative. A blood Wassermann was negative. The patient was put to bed and it was noted that he was drowsy a good deal of the time and his responses were slow. He was poorly orientated, irritable at times, and his insight was poor. Most of the time his train of thought showed marked interruption. His blood examination showed hæmoglobin 80 per cent., total red cells 3,500,000.

On January 29 a radiograph by Doctor Gray revealed a calcified

FIG. 8



Case V. Showing a pineal tumor involving the corpus callosum from the medial aspect of the brain.

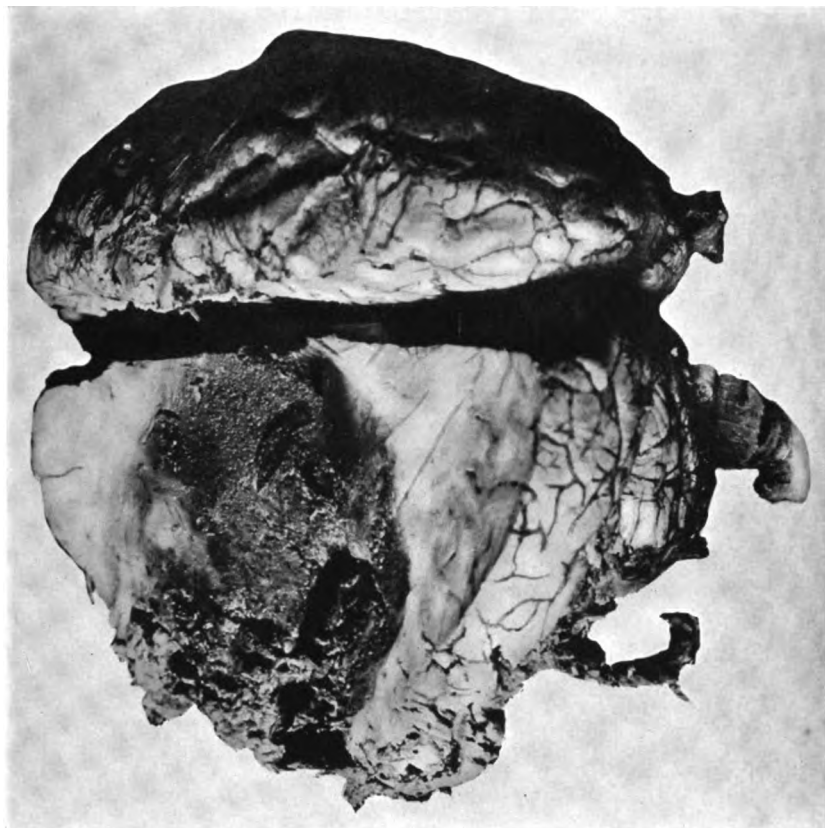


FIG. 10

Case VIII. Large left frontal tumor in the motor area. This tumor also involved the opposite hemisphere to some extent.

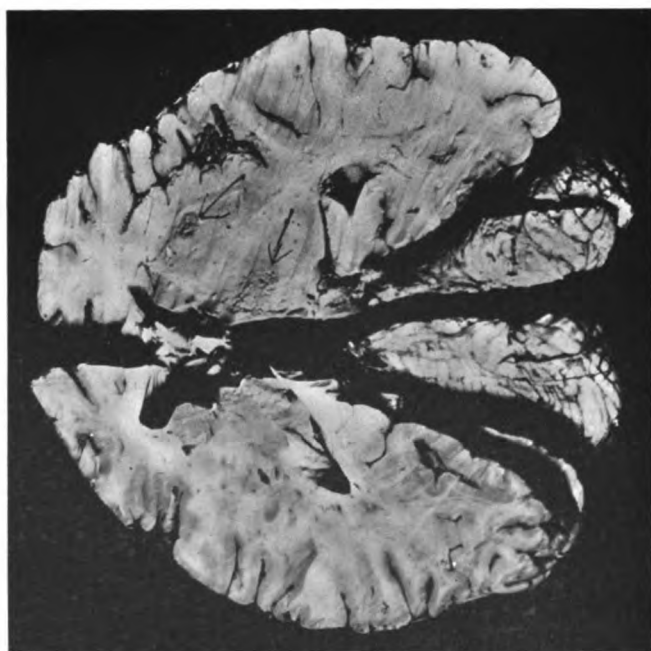


FIG. 9

Case VII. Multiple metastatic carcinomata of the brain. This section shows two growths in right hemisphere. Some twenty of these small tumors were found in various locations in both hemispheres.

mass slightly to the left of the mid-brain area. There were also radiographic evidences of increased intracranial pressure, manifested by thinning of the cranium and irregular indentations of the inner table, especially in the occipital region. The pituitary fossa was normal.

The spinal fluid gave a negative Wassermann, 22 cells to the cubic millimetre, and a faintly positive globulin. The fluid was under somewhat increased pressure. There were no signs of choked disks or optic atrophy.

The diagnosis of a brain tumor was made. Judging by the calcified area seen in the radiographs, it was deemed to be situated near the centre of the brain, slightly below the centre and to the left, and in the neighborhood of the posterior part of the corpus callosum.

On February 24, 1916, Doctor Frazier, of Philadelphia, operated, performing a callosal puncture. The right ventricle was found dilated and 10 Cc. of fluid evacuated. The pia-arachnoid was noted to be opaque in appearance. This puncture temporarily relieved the patient, except his mental confusion. From the time of this puncture the patient never complained of headache again.

He returned to the Tucker Sanatorium March 18, 1916. It was found that his memory was exceedingly poor. He was in a stupor most of the time, and it was hard to arouse him. His pupils were active and about equal. His extra-ocular movements were good and well sustained. There was no nystagmus or ptosis, and his fundi were negative. The slight right facial weakness remained. The tongue protruded in the middle line. There was no tremor or ataxia. The knee-jerks were increased on both sides. There was a well-sustained clonus on the right. There was no Babinski on either side. On March 29 the patient was very drowsy and had a pulse-rate of 135 and respiration of 40. A lumbar puncture was performed and 20 Cc. of clear fluid was withdrawn. After this the patient brightened temporarily. There were no cells in this fluid, but a marked excess of globulin.

The patient died April 12, and permission for an examination of the brain was obtained. A tumor measuring about $2\frac{1}{2}$ inches from anterior to posterior and 2 inches from side to side was found. The brain was hardened and the situation of the tumor was found to be chiefly in the left hemisphere, but markedly invading the right. It

pressed forward, denting the optic thalamus. It was attached to and involved the under surface of the corpus callosum, and also involved the commissural fibres between the middle and posterior commissures and slightly infiltrated the mesial aspect of the lenticular nucleus. The origin of the tumor was evidently in the pineal body on the left side. Pathologic examination proved the tumor to be a psammoma.

Remarks.—Here was a man seen by three neurologists in different cities, including myself, six months before he died of brain tumor and diagnoses of bilateral facial neuralgia, toxæmia, and drug habit were made. Several weeks after this the author saw him and changed his diagnosis from neuralgia to brain tumor. Bilateral neuralgias involving chiefly one and the same nerve on each side are usually due to a central lesion. For instance, bilateral sciatica is usually due to cauda equina tumor. The radiograph showed calcification in the suspected area and should have brought to mind a pineal growth, as calcification is not infrequently found in these cases. The radiograph also showed evidence of increased brain pressure. Bell⁷ says but few lesions of the pineal body have been observed. The most frequent symptoms of tumor of the pineal body are headache, vomiting, and optic neuritis, with languor and sleepiness. Ocular palsies, vertigo, staggering, tremors, deafness, pain, and rigidity in the neck have also been observed. He states that pineal tumors have occurred with few exceptions during childhood or early adult life (Fig. 8).

Although the Wassermann was negative, the fluid gave 22 cells per Cm. at first and slightly increased globulin, and afterward a marked increase in globulin.

It is to be noted that this case never had intra-ocular symptoms and his blood-pressure was normal. This case occurring in an adult showed no increased body or sex development.

This tumor indented the optic thalamus, but he had no thalamus syndrome, which, Ewerts⁸ states, according to studies by Roussy and by Holmes, consist mainly of hemi-anæsthesia, excitant hyperkinesis, hemi-loss of deep sensibility, slight hemi-ataxia, astereognosis, and severe subjective pain on the affected side. Slight hemiplegia, choreic

⁷ BELL, HOWARD H.: "Hyperplasia of the Pineal Body," *Journal of Nervous and Mental Diseases*, December, 1916, vol. 44, No. 6, p. 481.

⁸ EVARTS, ABRAHAM B.: "A Comparative Study of Cases Showing Thalamic Lesions at Autopsy," *Ibid.*, May, 1917, vol. 45, No. 5, p. 385.

and athetoid movements also occur. It also seems to have some connection with the emotions.

According to Bell, the pineal body reaches its maximum growth about the sixth year of life, and at the fourteenth year involution takes place. The pineal cells appear to be modified neurologic cells, with normal cells in islands nearly surrounded, as a rule, by septæ.

CASE VI.—*A Right Subcortical Temporal Tumor Resembling Cerebral Abscess.*

Male, age forty-seven, married, three children well, one feeble-minded. Past history negative, except he was knocked unconscious for twelve hours when twelve years of age. No scar was found. He was a commercial salesman and came under observation in November, 1916. Six months previous he found he could not sell goods as well, because he had become dull and stupid. He was thought by his physician to have neurasthenia and was sent to the country. He did not improve, and about a month before he came under observation he began to have vomiting, vertigo, and headache. His hearing was noted to be better at times than at others. There was no history of running ear. He had never been unconscious nor had convulsions.

Examination revealed absent knee-jerks on the right, but present and active on the left. There was clonus on the left, none on the right. There was questionable Babinski on the left, none on the right. His muscle power was weaker on the spastic or left side. His abdominal reflexes were absent. His elbow-jerks were normal. There were no tremors. The patient refused to talk. The blood-pressure was 180 systolic.

His right pupil was larger than the left and there was double choked disk. His cranial nerves could not otherwise be tested because of gradually deepening coma. He had incontinence of both urine and fæces.

His temperature varied from 98° to 100.6°, and his pulse from 50 to 90. His leucocytes were 11,600 and his polymorphonuclears 76 per cent. A Wassermann reaction on the blood was negative, and one on the cerebrospinal fluid was also negative. The fluid showed 10 cells and globulin negative.

The diagnosis of a right temporal abscess was considered, but because of the length of time the symptoms had lasted it was discarded in favor of a right subtemporal tumor. In a few days a right decom-

pression was done and the brain found under great pressure, but no tumor was discovered. The patient was put to bed and the symptoms persisted. In three days the operative site was enlarged downward and also forward and the brain explored, but no evidence of tumor was found. The patient died the day after the second operation, and post-mortem examination revealed a temporal subcortical tumor $2\frac{1}{2}$ by 2 inches, on the right side, under the operative site. It was an infiltrating endothelioma.

Remarks.—The reflexes were increased on one side and absent on the other, and the tumor was found on the side opposite the spasticity. As an elevation of temperature occurred at times, the pulse was slow, and there was a moderate leucocytosis, the diagnosis of a cerebral abscess had to be considered.

There has been considerable discussion on the subject of differential diagnosis between brain tumor and brain abscess. Burr⁹ says: "Tumors and abscesses have in the past and will again in the future be mistaken for one another, and this is in the nature of things, for both are alike in that they are foreign bodies in the brain." And again: "The diagnosis of abscess depends much more upon the existence of symptoms outside the brain, and the finding of an adequate cause, than it does upon the nervous symptoms." Sharpe¹⁰ states that 60 per cent. of brain abscesses occur in the temporosphenoidal lobe. He calls attention to the periodicity of symptoms or intervals of remission of symptoms.

The diagnosis between tumor and abscess in the temporal region in the presence of increased temperature is certainly difficult, but, in the author's opinion, if the ears are normal and there is no history of running ear in the past, and the patient is suffering from no acute infectious disease, and no focal sign of pus can be found elsewhere in the body, the diagnosis is in favor of tumor.

CASE VII.—*Multiple Tumors, from Carcinomatous Metastases, of the Brain.*

Female, white, married, age fifty-five years. Was first seen January 11, 1917. The family history was negative. The past history

⁹ BURR, CHAS. W.: "A Case of Tumor of the Brain Mistaken for Abscess," *University Medical Magazine* (University of Pennsylvania), December, 1908.

¹⁰ SHARPE, WILLIAM: "The Diagnosis and Treatment of Brain Abscess," *The Laryngoscope*, March, 1914.

was negative, except that the left breast was removed for carcinoma nine months before. For three months previous to examination she had complained of right frontal headache, with vomiting and vertigo.

Her mental reactions were found retarded and she was confused. Her station was poor, her gait unsteady and her knee-jerks exaggerated. There was a slight Babinaki reflex on both sides, more marked on the left. There was no clonus. There was incoördination of the lower extremities. Her speech was indistinct. Her ocular accommodation was poor, while the pupils reacted to light sluggishly and the left pupil was dilated. There was no exophthalmos or nystagmus. She complained of diplopia. An intra-ocular examination was negative except for congested veins. There was bilateral paresis of the third and fourth nerves. Her other cranial nerves were negative. Her pulse was normal and her blood-pressure was 142 systolic.

Both blood and spinal fluid Wassermann reactions were negative. There were no cells in the fluid, and the globulin was negative. Examination of urine was negative but for a trace of albumin and occasional cast.

The diagnosis of a carcinomatous tumor of the brain, unlocalizable, was made. The patient died in about a month, and an autopsy revealed a brain with multiple metastatic growths from a quarter to three-quarters of an inch in diameter. Microscopic examination proved these growths to be endotheliomata. There were many of these growths in both hemispheres and in various locations (Fig. 9).

Remarks.—It happens to be my experience in every case of brain or spinal lesion occurring in a person who had ever been operated upon for cancer, or who had a known cancer, that the lesion of the central nervous system turned out also to be carcinomatous. Of course, this is not invariably so, but in these cases metastasis is a safe guess. As this patient had multiple growths in both hemispheres, localization was impossible. Multiple growths should have been thought of in this instance, but they were not. The bilateral involvement of the third and fourth nerves and bilateral Babinski reflex should have indicated involvement of both hemispheres. It is noteworthy that there was no optic neuritis or choked disk.

Leving¹¹ thinks that metastasis of carcinoma or of sarcoma in the

¹¹ LEVING, ISAAO: "Metastasis of Cancer in the Central Nervous System," *Journal of Nervous and Mental Diseases*, June, 1917, vol. 45, No. 6, p. 48.

brain is a comparatively rare finding, and in this I agree, but my experience is that this is not true of the spinal cord. Leving also says that carcinomas spread by blood-vessels as well as the lymphatics, and makes the interesting observation that metastasis to the central nervous system may give no clinical symptom during its course, because it grows by replacing and destroying tissue, so that the combined amount of tissue in the skull may not change. This may be true in so far as the general signs of increased intracranial pressure are concerned, and it may be true also in gliomas, but the destroyed brain tissue usually gives some paralytic, mental, or cranial nerve evidence of its presence.

CASE VIII.—*A Large Left Frontal Tumor of the Motor Area Involving the Opposite Hemisphere in a Patient with Atrophy of the Right Arm and Shoulder.*

Male, married, age thirty-seven, teacher, white. First seen May 27, 1916. The family history was negative. The past history showed no serious illnesses except malaria in 1912. The patient was well up to five weeks previous to examination, when he began to be gradually paralyzed on the right side, the paralysis involving the face, first, then the arm, and later the leg. He had no nausea, vomiting, vertigo, or headache. He complained of numbness of the right thumb and some of the right forearm.

His general examination was negative, except his pulse was slightly rapid and his complexion was sallow. Urinalysis was negative. His blood-pressure was 124 systolic and 82 diastolic. His perspiration was normal. His blood was morphologically negative, and negative for malaria and lead. His blood Wassermann reaction was also negative.

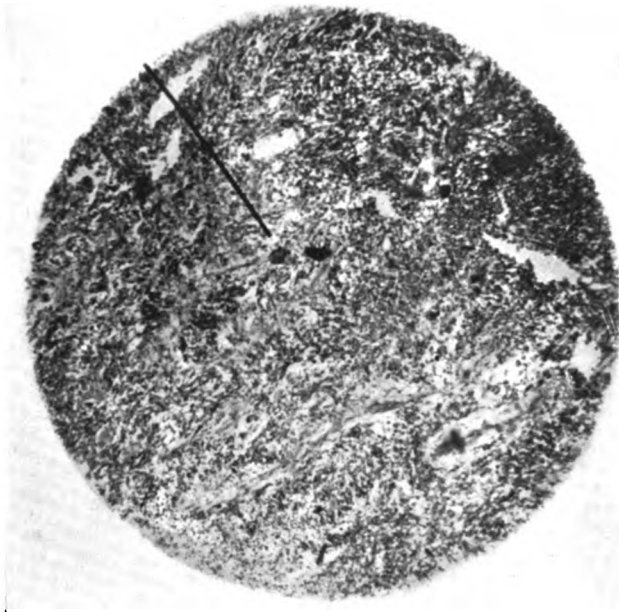
Neurologic examination showed partial motor loss in right face, arm, and, to a less degree, in the leg. His gait was slightly hemiplegic. He had some fibrillary twitching of the muscles of the right side of the body. There was a questionable Babinski reflex on the right, but none on the left. There was no clonus, and the knee-jerks were normal. In the right hand there was considerable atrophy affecting the thenar and hypothenar muscles, and to some extent in the shoulder muscles, especially the trapezius. The right arm was ataxic. Electrical examination of the affected muscles of the right arm showed faradic hyperexcitability and partial reaction of degeneration. A fine hand-tremor was present on both sides. Sensation to touch, pain, heat, and cold

FIG. 11



Case VIII. Showing the invasion of the right hemisphere in a large left post-frontal tumor of the left hemisphere.

FIG. 12



Case IX. Photomicrograph of an endothelioma of the temposphenoidal lobe. Note lymph-spaces and arrangement of endothelial cells forming around spaces and vessels, also strings of fibrous tissue.

was everywhere normal. He had very slight aphasia and his speech was rather mumbling.

The pupils were normal in size and shape and reacted to light and accommodation. There was slight nystagmus to the right, but no diplopia, exophthalmos, or ptosis. The margins of both disks were somewhat blurred. There was paresis of the right side of the face, nerve-deafness on the right, and the tongue protruded to the right.

Mental examination was negative except for indefinite lapses of memory and the fact that he recently became more easily excited than usual.

Examination of his cerebrospinal fluid showed the fluid under slightly increased pressure, Wassermann reaction negative, no cells, and globulin not increased. It was noted that on the scalp over about the location of the arm centre of the left motor cortex there was a scar which the patient believed had been present from birth.

The diagnosis of a cyst of the left hemisphere involving the motor area was made.

The patient was operated on June 26, 1916, and no tumor was found. The surgeon and a consulting neurologist formed the opinion that his "symptoms were due more to a vascular disturbance, an obliterative arteritis with secondary oedema, than to a subcortical growth; the latter, however, can not be altogether eliminated." The brain at operation was under moderate increased pressure.

The patient came home and became worse, and during the following September went to Johns Hopkins Hospital and was again operated upon over the previous site, and it was reported that "a large subcortical, infiltrating glioma was found invading about half of the left hemisphere." The tumor could not be removed, and the patient returned to his home and died in January, 1917.

An autopsy was performed and an infiltrating glioma was found as large as a small grape-fruit, occupying the middle three-fourths of the left hemisphere and extending over to and involving the right hemisphere (Figs. 10 and 11).

Remarks.—Here we have the case of an enormous brain tumor invading both hemispheres without nausea, vomiting, vertigo, headache, or actual choked disk. There was only some blurring of the margin of the disks. There were no distinct organic reflex changes. There was atrophy of the right arm and shoulder with

fibrillary tremor, electrical changes, and motor weakness, all indicating a lesion of the lower motor neuron type. The question which naturally arises is, Upon what was the diagnosis of brain tumor founded? In answer we may say that the diagnosis of brain tumor was made because of the gradually extending paralysis of the right side of the body, the slight aphasia, the blurring of the margin of the disks, the questionable Babinski, the slight nystagmus to the right, the nerve-deafness on the right, the tongue protruding to the right, the cerebrospinal fluid being under slightly increased pressure, and the fact that there was a scar on the scalp over the left motor area. The explanation of the atrophy with fibrillary twitching and electrical changes must lie between a separate lesion or a secondary trophic influence due to the cerebral neoplasm. Unfortunately the cord could not be examined.

The patient was not seen the last few weeks of his life, and it was not ascertained whether his motor symptoms became diplegic or not, but from the amount of invasion of the opposite hemisphere it is assumed that they must have done so.

CASE IX.—A Right Temporosphenoidal Tumor of Very Rapid Development, Which Blocked the Sylvian Aqueduct, and Extended into the Occipital Lobe.

Male, white, mill hand, age twenty-six, was first seen December 18, 1916. His family and past history were unimportant. He had complained of a pain in the right side of the head, running down into the face, for a little over a month. He also had some nausea, and twice since this pain began he vomited without cause. He also complained of vertigo, and stated that when the headache came on he had an imaginary smell of some strong medicine in the right nostril only. There were no other subjective sensations.

His neurologic examination showed a slight Babinski on the right side before a lumbar puncture was performed, but none after. He also had a suspicion of clonus before the lumbar puncture, but none afterward. There was no paralysis and no ataxia. His muscle power and muscle tone were good. His knee, Achilles and elbow-jerks were active and equal, and his superficial reflexes were active. There was no tongue- or face-tremor, but a fine hand-tremor. His speech was negative, except that he was rather deliberate in articulation. His sensation was normal. His pulse varied from 60 to 70.

His eye examination revealed double choked disks, greater on the right side. There was no ptosis, no nystagmus, no exophthalmos. Examination of his other cranial nerves was negative except for small hallucinations, pain over the right side of the face, and diminution of bone conduction in the right ear.

Mental examination showed all reactions normal except considerable mental hebetude.

A Wassermann test on the blood was negative, and a blood smear was also negative. The spinal fluid was under increased pressure and was negative for globulin, cells negative, Wassermann negative.

The diagnosis of deep-seated temporo-occipital tumor on the right side, affecting the uncinate gyrus, was made. The day after the examination the patient became comatose; on the next day, December 20, 1916, he was operated upon by Dr. C. C. Coleman, who performed a right subtemporal decompression and found great increase in intracranial pressure. The patient died on December 21.

Autopsy was allowed, and the brain showed considerable dilatation of the left lateral ventricle, the right lateral ventricle apparently containing no fluid. On section of the brain we found an infiltrating growth about $2\frac{1}{2}$ inches by $1\frac{1}{2}$ inches, extending from the cortex of the anterior part of the right occipital lobe downward and forward deeply into the region of the midbrain. The growth had blocked the Sylvian aqueduct and could not have been removed by operation. The tumor was reported by the pathologist to be an endothelioma (Fig. 12).

Remarks.—The localization of this growth in the temporo-occipital region involving the uncinate gyrus was made chiefly because of the presence of hallucinations of smell, coupled with the slight cranial nerve involvement and absence of other localizing signs. The growth, in reality, involved both the temporal and somewhat the occipital lobes. The hallucinations of smell were due to neighborhood pressure rather than direct pressure on the uncinate gyrus.

It is also worthy of note that a lumbar puncture may change organic signs—a fact to which I have seen no reference in the literature. This observation has also been made in other cases. In one instance a child with very diminished knee-jerks and a flexor plantar reflex developed, within twenty-four hours after the removal of 20 Cc. of fluid, active knee-jerks and a Babinski reflex on the left side. The fact that this growth blocked the Sylvian aqueduct probably

accounted for the rapid development of the symptoms, as well as the increased fluid in the left hemisphere. It should be noted that the dilated lateral ventricle was on the side opposite the lesion, and that the ventricle on that side was blocked.

It is interesting to note that facial pain in this case was a prominent feature, as it was in Case V, and was probably due to pressure-irritation on the gasserian ganglion.

✓ CASE X.—*A Tumor of the Left Pontocerebellar Angle, with Bilateral Loss of the Sense of Smell and Diminution of the Sense of Taste.*

Female, single, twenty-three years of age, seen in April, 1911.¹² The patient never had a serious illness until whooping-cough four years before, after which she began to have severe pain in the back of the neck and nausea. During the previous year this pain had been more severe and was accompanied by vomiting. There were short periods of loss of vision during exacerbations of the headache. The patient had always been more or less quick-tempered and emotional, and had alternations of depression and of elevation of spirits.

When questioned she complained of pain at the junction of the neck and skull, of coccygodynia, of aching in the limbs, of a queer feeling in the left ear, of periods of blindness lasting a minute or so, of trembling in the left arm, of disturbance of the sense of taste and smell, of slight difficulty in walking, of lost energy, of vertigo, of poor sleep, of variable memory, and of constipated bowels. She had never had convulsions or unconsciousness.

Upon examination it was found that her pupils were dilated, but reacted fairly well to light and accommodation; that she had nystagmus in all directions; that her vision fields were roughly good; that both optic nerves showed evidence of neuritis with beginning atrophy, and that there was no hemianopia. There was loss of smell on both sides, great diminution in the sense of taste, and paralysis of the left recurrent laryngeal nerve. She was deaf in the left ear, but could hear well in the right. When a pencil was put in the left hand between the fingers her forearm would go into a clonic rotary spasm and the pencil was thrown out. The left nasolabial fold was greater than the right. She held her head inclined slightly to the left shoulder. Her

¹² TUCKER, BEVERLEY R.: *Old Dominion Journal of Medicine and Surgery*, vol. xiii, No. 6, December, 1911.

tongue protruded straight, and sensation to touch over the face was normal. Sensation of the pharynx on the left side was almost completely lost, but was normal on the right side. Motion of the soft palate was good on both sides, articulation was good, and there was no drawing, but some hoarseness of speech.

Her knee-jerks were slightly exaggerated, but equal on both sides; her station was unsteady with eyes opened or closed, and she staggered to either side in walking. Turning quickly gave her vertigo. She was unable to stand on one foot at a time. Her grip was normal in each hand. Sensation to touch was good. There was no astereognosis. There was incoördination of both upper extremities. There was slight attempt at ankle clonus on the right side, but there was no Babinski sign on either side. Her blood-pressure was 118, pulse 100, and temperature normal. Her urine was examined and found to be normal, except for an excess of phosphates.

The first, second, fifth, seventh, eighth and ninth, and possibly the tenth, nerves were more or less affected.

The diagnosis of a tumor of the pontocerebellar angle on the left side was made, and on May 1, 1911, she was operated on by Dr. Harvey Cushing, who wrote: "It was an unusually difficult case, owing to a very high-lying lesion. The growth was the usual circumscribed endothelioma of the angle, and, as they are benign, the mere leaving of some fragments of the tumor *in situ* does not necessarily mean that there will be any prompt recurrence of symptoms, although it is possible that a second performance may be necessitated in time."

January 16, 1917, nearly six years later, the following letter was received from her father: "Her sight, hearing, and sense of taste and smell are all gone on the left side. She sees badly out of the right eye, but can read very large print. She has no headaches and her general health is good. She has never been able to stand alone, but walks from room to room with support. She can not lift a glass of water with her left hand or step surely with her left foot."

Remarks.—It is not infrequent for a brain tumor to follow an acute infectious disease, just as it is not uncommon for one to follow a blow on the head. In this case the symptoms were dated back to whooping-cough. Her symptoms at first somewhat resembled migraine, except the pain was occipital, bilateral, and occurred in exacerbations rather than in periodic attacks.

This patient also gave a history of cyclothymia (manic-depressive psychosis), but this antedated, and was evidently independent of, her brain tumor.

A remarkable fact in this case is that there were bilateral loss of the sense of smell and bilateral diminution of the sense of taste. The sense of smell returned after the operation. The author can not account for these symptoms occurring in a cerebellar pontile growth. It is also worthy to note that the sixth nerve was not affected.

The multiple cranial nerve involvement ruled out a strictly cerebellar growth. Weisenberg and Work¹⁸ have stated that "the presence of cranial nerve symptoms, as the seventh, sixth, fifth, ninth, tenth, or twelfth, indicates that the lesion is extracerebellar."

An operation, which at least gave her relief from pain and nausea, returned her sense of smell, and preserved her intelligence, was highly justifiable.

CASE XI.—A Right Subcortical Cystic Tumor Resembling Cortical Hemorrhage, Involving the Motor and Sensory Areas, Operated on with Recovery.

White, male, age nineteen, pupil, seen June 22, 1915. Family history negative. The past history revealed that he was thrown from a horse when four years old and was unconscious for one-half hour.

The first he knew of his present trouble was three weeks before he came under observation, when it was noted that he gradually lost power in the left side of his face, then his left arm, and in a week his left leg. For two weeks he had severe headache and complained of failing sight. There was no nausea, vomiting, or vertigo.

Examination revealed moderate optic neuritis and possibly some atrophy, no exophthalmos, weakness of the seventh and twelfth nerves, slight paresis of the left third and lessened sensation in the distribution of the left fifth. The patient also had a Babinski on the left and general sensory and motor loss on the left face and left arm and leg. There was no aphasia. The left knee-jerk was exaggerated. His gait was hemiplegic. His sphincters were normal.

Mental examination showed only hebetude and poor memory.

¹⁸ WEISENBERG, T. H., and WORK, P.: "The Diagnosis of Tumors in the Posterior Cerebral Fossa," *Journal of the American Medical Association*, October 16, 1915, vol. 65, p. 1345.

His blood-pressure was 130 systolic and 80 diastolic. Examination of his blood was negative. His cerebrospinal fluid was under greatly increased pressure, but the Wassermann, cell count, and globulin were negative.

The diagnosis of a rapidly growing tumor in the right hemisphere, catching the motor and sensory radiation between the cerebral cortex and the internal capsule, was made.

The patient was operated upon by Dr. Chas. H. Frazier, July 2, 1915, who reported: "In accordance with the symptomatology, we opened the skull, to expose the right motor and parietal region, and there found the lesion, which, I believe, is a cystic glioma. The postcentral convolution was pale and of a yellowish color, and palpation over it detected fluctuation. Upon dividing the cortex, an underlying cyst, containing a considerable quantity of straw-colored fluid, was revealed. A portion of the cortex was removed for microscopic examination, and the fluid, also, for examination. The microscopic appearance was that of a cystic glioma." A later pathologic report confirmed the diagnosis.

The patient made a good recovery, and on examination October 27, 1915, the following notes were made: "Feels well, memory normal, sensation normal, slight weakness of right wrist, left knee-jerk slightly exaggerated, right normal, and there was no clonus or Babinski. Eye examination was negative, including normal disks. There was no ataxia, no tremors, no weakness of the face."

Reëxamination in October, 1916, was entirely negative, and the young man has been working steadily as clerk for over a year. September, 1917, the young man was still well.

Remarks.—This case began with gradual motor involvement of the left side of the body. The cranial nerve symptoms came on rapidly, and the patient was operated upon and the growth removed within five weeks after the first symptoms were manifested. Although the growth was rapid and the cerebrospinal pressure increased, there was no vomiting or vertigo and the blood-pressure was normal. His recovery was prompt and complete.

The onset of this case made one think of a cortical hemorrhage, for within less than two weeks he was markedly hemiplegic. However, the normal blood-pressure, negative Wassermann, and absence of history of immediate injury were against the diagnosis of hemorrhage.

CASE XII.—*A Tumor of the Pontocerebellar Angle with but Few Cranial Nerve Symptoms, Attached to the Pons and Indenting the Under Surface of the Cerebellum.*

White, male, age thirteen, was first seen October 29, 1913. The family history was negative. The past history was negative, except that a year previous to examination this patient had a fall from a horizontal bar, struck the back of his head, and was dazed for a few minutes. Six weeks before he came under observation his adenoids and tonsils were removed, and this operation was followed in a few days by paralysis of the right external rectus, and this, in turn, in a week, by paresis of the left external rectus. Shortly afterward he began to have nausea, vomiting, vertigo, and frontal headache. There were also numbness and tingling of the left arm and leg. His temperature and pulse were normal.

Examination revealed poor station with lurching to the left, and unsteady gait with lurching also to the left. The knee-jerks were exaggerated, but greater on the left. The elbow-jerks were exaggerated. The abdominal reflexes were diminished. Babinski and clonus were present, both more marked on the left. There were marked ataxia and incoördination. There were no tremors. Speech and sensation were normal. The patient was myopic. His pupils reacted to light and accommodation, he could not converge, and he had slight ptosis, more marked on the left. Marked nystagmus was present in the left. His fundi were negative. His eyes were examined again just before operation by Doctor Holloway, of Philadelphia, who reported he had no papilloedema. There was no exophthalmos and no hemianopia. His cranial nerves were negative, except for weakness of both external recti, ptosis, and eighth nerve deafness on the left.

Examination of the urine was negative. A differential blood count was negative. A Wassermann reaction, globulin estimation, and cell count on the cerebrospinal fluid were negative. The fluid was under increased pressure. His mentality appeared entirely normal. A radiograph of his skull was negative. His blood-pressure was normal.

The diagnosis of a tumor in or near the pontocerebellar angle was made. He was thought by his physician to have some form of meningitis, and this diagnosis was thought more probable than a tumor by a consultant from a distance.

On November 10, 1913, he was operated on by Dr. Charles H.

Frazier and an attempt was made to expose the pontocerebellar angle. The boy's condition became bad and the operation had to be discontinued. The patient died the next day. The following is the post-mortem report:

"By dividing the cord just below the medulla, we were able to displace the hemisphere sufficiently to secure an excellent exposure of the cerebellar pontile space, and here a tumor was found of considerable dimensions, of jelly-like consistency, and of a dead-white color, precisely like that of the white matter of the brain, beginning at its outer margin about the level of the posterior lacerated foramen, where it passed directly on the ninth, tenth, and eleventh nerves, which leave the skull at that point. From this point it extended inward and was attached to the pons. The seventh and eighth nerves passed over and were put on the stretch by the tumor. The tumor had made by pressure a depression on the surface of the cerebellum immediately adjacent to it. It had not infiltrated either cerebellar hemispheres. Line of demarcation could be clearly seen between the two. One could sweep the finger around the tumor on all sides except the point at which it seemed firmly attached to the pons. From the surgical point of view the tumor would be classified as inoperable. Even at the post-mortem examination it could not be removed in its entirety, but only fragmentarily, on account of its soft, jelly-like consistency and absence of a definite capsule."

Microscopic examination showed the tumor to be a glioma. Pathologic report from the University of Pennsylvania Hospital is as follows: "The size of the part of the tumor removed was $4 \times 3 \times 2$ Cm. Microscopically, the tissue is seen to consist of a fine-fibrillar stroma containing large numbers of round or oval cells with clear-cut, deeply staining nuclei and comparatively little cytoplasm. In addition, there are scattered here and there a few much larger cells having a good-sized nuclei and large, somewhat stellate or irregular cell body which takes the eosin stain. Throughout the stroma are large capillary vessels and a few areas of hemorrhage."

Remarks.—As this tumor was attached to the pons, it is fair to assume that it originated from pontine tissue. It is important to note that choked disk never appeared. In fact, the cranial nerve symptoms in this case were confined to two, the sixth and the eighth, although at post-mortem examination the tumor was noted to rest on the ninth,

tenth, and eleventh nerves and that the seventh and eighth were stretched over it. The fact of the slight cranial nerve involvement made the diagnosis difficult. Another confusing symptom was that the headache was frontal. The ataxia and nystagmus, and sixth and eighth nerve paresis, however, were thought sufficient to put the localization in the pontocerebellar angle. The symptoms were rapid in development and seemed to have some relation to a blow on the head a year before.

Just what relation, if any, the operation for removal of the tonsils and adenoids had to this case it is impossible to say.

CASE XIII.—*A Tumor of the Right Pontocerebellar Angle Which Had Been Mistaken for the Cerebellobulbar Type of Poliomyelitis.*

Seen June 27, 1916, white, age five and one-half years. The family and past history were negative.

This boy was always in good health until about eight weeks before he came under observation, when he fell from a sofa and struck his head on a toy iron train of cars. There was no scar on the head to show the injury. This fall was followed by an attack of vomiting and other digestive disturbances, and from time to time since he had had general headache and vomiting. Sometimes this vomiting was thought to be due to his food or medicine, at other times it was without cause. He had no vertigo. About two or three weeks after the accident it was noticed that his eyes were not straight, that he had some difficulty in walking, and that the right side of his face drooped.

At the time of the first examination he had right facial palsy, decided paresis of the right sixth nerve, poor station, questionable Babinski on the left, slight ataxia in gait, no clonus, exaggerated knee- and elbow-jerks, diplopia, and some nystagmoid jerkings, but no true nystagmus.

He was seen again on June 30 and the Babinski sign was prominent on the left and there was an attempt at clonus on the left. There was no astereognosis. His gait had not improved. His ears were examined by Doctor White, who reported that there was no eighth nerve involvement. His fundi were normal except for some venous congestion.

He was seen again on July 7. The gait was decidedly cerebellar in character and there was more marked ataxia. He had vomited

once or twice without cause. No vertigo had developed. His station was worse and he had developed considerable deafness in the right ear. The motor portion of the fifth nerve was slightly weak on the right.

Wassermann reaction was negative on the blood and cerebrospinal fluid. A cell count and globulin were also negative on the cerebrospinal fluid.

In view of the history of headache and vomiting, the involvement of the fifth, sixth, seventh, and eighth nerves on the right, and his ataxia, a diagnosis of a right-sided pontocerebellar angle growth was made.

The boy was sent to Philadelphia for operation, but the consulting neurologist did not agree in the diagnosis and wrote, July 12, 1916: "The question naturally arising in this case is as to whether he has a cerebellopontile neoplasm or some other form of destructive disease involving his cerebellum, pons, and oblongata. His present condition seems to have been ushered in by an acute attack some six weeks ago. This had the features of an acidosis with constipation, vomiting, etc., and probably some fever, but I could not get any record of this. My own conviction is that the child had about this time an attack of acute poliomyelitis of the cerebellobulbar type—and a favorable prognosis was given."

Later the diagnosis of a pontocerebellar angle growth was concurred in, especially after a Barány test, and the child was operated on August 8, 1916. The operative notes were as follows: "A suboccipital craniectomy was done on the right side. Upon opening the dura the cerebellum bulged considerably. Examination of the right cerebellopontile angle showed that there was probably a tumor over which certain of the nerves were stretched. The view was not entirely clear and I deemed it inadvisable to go further. Accordingly the external wound was closed in the usual manner." The consulting neurologist wrote that he had changed his diagnosis to pontocerebellar angle tumor, and that he had seen the tumor in this location at the operation.

Another operative attempt was made August 21, 1916, but nothing definite was shown except the signs of increased intracranial pressure.

The child returned to Richmond and gradually grew worse. A cerebral hernia appeared in the right suboccipital region about three inches in diameter and elevated about an inch. Vomiting occurred

repeatedly, headache was severe, vertigo never appeared, the disks remained clear, the paralytic symptoms and ataxia noted before the operation persisted, but with greater intensity. On November 29, 1916, he died, evidently from a cerebellopontile tumor. Unfortunately an autopsy was refused.

Remarks.—This was a much-discussed case. The rapid development of symptoms, the fact that the patient had acidosis and a stomach upset, and that his illness occurred during the epidemic of poliomyelitis in New York led several eminent physicians to believe that the case was one of the cerebellobulbar type of poliomyelitis.

Among the reasons why the diagnosis of pontocerebellar angle tumor was adhered to by us were the persistent headache and vomiting, the fact that the cranial nerves became involved one after another, and that the boy became progressively worse instead of better, as he probably would have done in poliomyelitis. In poliomyelitis the maximum of damage is done at first and there is a tendency toward improvement.

This is another case in which there were no signs of optic nerve involvement even to the end. The history of a blow on the head at the onset of the symptoms is also interesting.

CASE XIV.—A Tumor of the Right Postcentral Frontal Region Resembling a Cerebral Abscess.

Age fifty-six, married, had six children, was seen first May 28, 1917.

The patient was referred as a case of paresis. His family history was negative. His past history showed nothing of importance except the history of possible grippe five months previous, and since that he had a slight afternoon temperature. He had no history of running ear, and a radiograph gave no sign of middle-ear trouble.

The patient had been constipated and had lost ten pounds in three weeks, although his appetite was voracious. There was no nausea, vomiting, vertigo, or headache. His sleep had been poor. Examination showed that he had an occasional clonic trembling of the left leg. He also had some tongue-tremor. His knee-jerks were exaggerated. He had a double Babinski, equal on both sides, but no clonus and no ataxia. His elbow-jerks were normal. He had considerable general muscular rigidity. His pupils were sluggish, but reacted to light and were normal in shape and size. There was no nystagmus,

exophthalmos, or ptosis. His pronunciation was normal, but his speech was slow and he showed a tendency to repeat what he said. His memory was poor and he was nervous and confused.

Examination of his eyes by Doctor Wright revealed blurring of the nasal half of both disks. Examination of the urine was negative. An X-ray examination of his skull showed that the pituitary fossa was flattened, but there was nothing to indicate the presence of abscess or tumor.

The blood-pressure averaged about 150. The cerebrospinal fluid was under considerably increased pressure. The cell count, globulin, and Wassermann reaction on this fluid were negative. A Wassermann on his blood was also negative. Examination of his blood showed 8000 leucocytes, a negative smear for malaria, and polymorphonuclears 80 per cent. His pulse-rate was between 80 and 100, and his temperature between 98 and 100.5° F.

The diagnosis was made of either an abscess or a tumor in the right temporo-phenoidal region. The patient was operated on by Dr. C. C. Coleman on June 10, 1917, who performed a right sub-temporal decompression and found his brain under very considerable pressure. The veins were distended and the dura looked darker than usual. Upon opening the dura the brain herniated. The brain was needled, but no abscess was found, nor was there any increased resistance which might indicate a tumor.

The patient died several days after the operation, and autopsy revealed a tumor in the right frontal region just posterior to the centre. This growth was about 1½ inches in diameter, soft in consistency, and was a glioma.

Remarks.—This case presents many features of considerable interest. In the first place, the tongue- and face-tremor, mental confusion, and increase in muscle-tone looked something like paresis. The question of paresis, however, was discarded at the first examination and he was not seriolgically luetic.

The fact that the patient had a marked tremor of the left leg and slight tremors elsewhere, with muscular rigidity, made one think of paralysis agitans. This, however, was discarded as soon as the presence of organic symptoms like the Babiniski sign and eye changes were found.

The fact that he had daily elevation of temperature and the history

of the symptoms beginning with some acute infectious condition, thought by his wife to be grippe, combined with his brain-pressure symptoms, made brain abscess a consideration.

The diagnosis then lay between a cerebral abscess and cerebral tumor, with the possibilities in favor of abscess. The location was thought to be temporo-phenoidal because of the few reflex and the paralytic changes present. A frontal growth, however, should have been thought of in view of the mental symptoms and the increase in muscle-tone, to which Mills recently called attention.

This case is also interesting because of the fact that there was no nausea, vomiting, vertigo, headache, or true choked disk present at any time during the course of the condition. His fundi showed only a blurring of the nasal halves of the disks. His temperature may have been due to some low-grade pulmonary affection. Unfortunately permission could be obtained only to make a post-mortem examination of the head.

CASE XV.—A Tumor of the Superior Surface of the Cerebellum in Which the Diagnosis of Acute Hydrocephalus Had to be Excluded.

White, male, age eight years, first seen June 17, 1917. The family history was negative, except that he lost one brother three days old, cause unknown, and one sister six months old, cause unknown.

The past history was negative, except for diphtheria when two years of age.

The present illness, so far as parents knew, began three weeks before the patient came under observation. The child was playing and his head became caught in a well top. The blow was not violent, but there was considerable pressure on the back part of the head for a few moments. After this he complained of pain in the back part of the head, and after about a week began to vomit at intervals. In a few days after he had vertigo and malaise, and in about a week more he went to bed, chiefly because of increased vertigo, vomiting, and drowsiness.

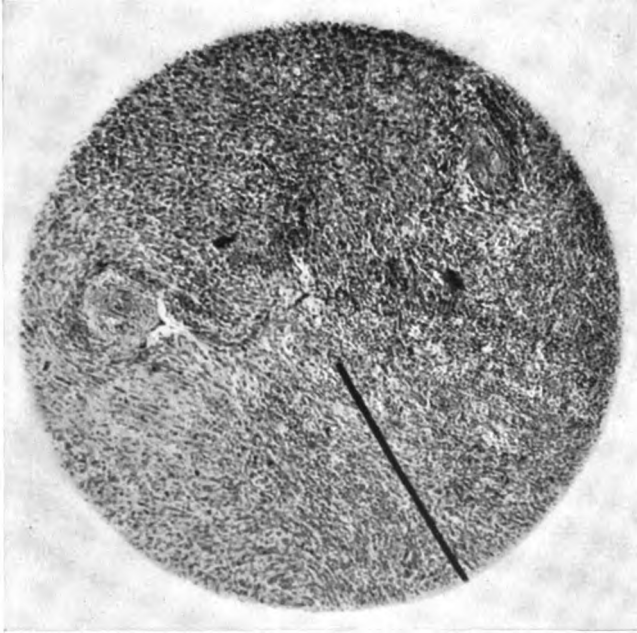
Upon examination the boy was found to be well nourished, and his gait was unsteady, but not otherwise peculiar. He had some stomatitis. The child's head was large and the forehead prominent, but this was said to be a family characteristic. His superficial reflexes were normal, his knee-jerks were diminished, there was no clonus and no Babinski, but slight double Kernig's sign was present. The

Fig. 13



Case XV. Showing a gliosarcoma on the superior surface of the cerebellum. The symptoms came on rapidly following an injury.

FIG. 14



Case XV. Photomicrograph of a gliosarcoma on the superior surface of the cerebellum. Note round and spindle-shaped cells, irregularly shaped nuclei, and mitotic figures in large numbers. Vascular stroma to left and in right upper segment.

abdominal muscles were a little rigid. There was some ataxia and "overreaching" of both the left upper and lower extremities. The tongue protruded slightly to the left, which, except for his slight optic nerve involvement, constituted his only cranial nerve symptoms. There was considerable hypersensitiveness of the feet and also of the deep muscles of the legs and thighs. There was no increase of muscle-tone. The spine was straight and not rigid.

The pupils reacted to light and accommodation, and there was no nystagmus, ptosis, diplopia, or exophthalmos. An intra-ocular examination by Doctor Bowen showed a number of small hemorrhages in the retinae of both eyes, slight papillitis, and veins which were enlarged and tortuous.

Blood examination showed 8100 leucocytes and 81 per cent. polymorphonuclears. The urinalysis was negative. A lumbar puncture was performed and 22 Cc. of fluid under pressure was withdrawn. This fluid gave a negative Wassermann reaction and also was negative for globulin and cell count. It was also bacteriologically negative.

After the lumbar puncture Babiniski sign appeared on the left side for a few days, then disappeared. The mental condition of the child brightened up so much after this puncture that the parents thought he was getting well. In a few days, however, drowsiness, vertigo, and vomiting returned.

A week after he came under observation Dr. C. C. Coleman performed a right subtemporal decompression. He found the brain congested and the dura tense, and he relieved the patient of a great quantity of cerebrospinal fluid. This decompression was done preliminary to an occipital operation which was to have been performed in about a week.

The boy did well for thirty-six hours after operation and then died suddenly of medullary failure, which was thought to be due to a shift in the position of the tumor.

At autopsy a large gliosarcoma, $2\frac{1}{2} \times 2 \times 1\frac{1}{2}$ inches, was found on the posterior-superior aspect of the cerebellum, attached to and slightly invading the cerebellar cortex, especially on the right (Fig. 13).

Remarks.—This case is of interest mainly because the boy was a healthy country lad who showed no symptoms of any illness until after a slight injury to the back of the head just one month previous to his death. This might make one expect to find an hæmatoma, but

instead there was a large, soft, white gliosarcoma with no evidence of hemorrhagic origin (Fig. 14).

One of the consultants thought that the diagnosis was acute hydrocephalus, as described by Oppenheim and others. The fact that his head was larger than normal and his forehead more prominent was thought by him to be a predisposing factor to an acute traumatic hydrocephalus from his blow on the back of the head. However, the diagnosis of a cerebellar growth was persisted in.

GENERAL DISCUSSION OF THIS GROUP OF CASES

In the consideration of some of the symptoms occurring in brain tumors it might be well to call attention to a few recent observations which have been made. Weisenberg¹⁴ states that irritation of the roots of the glossopharyngeus may simulate tic-douloureux. In several of these cases pain in the face was a prominent feature, but the author attributes it, in these instances, rather to fifth-nerve irritation than to glossopharyngeal. Because choked disk may occur in chronic nephritis this condition may be confused at times with brain tumor. The author has no case in point in this series. Exophthalmos may occur from brain tumor, but there was only one of these cases (Case I) in which exophthalmos was present. Frazier,¹⁵ in speaking of hydrocephalus in comparison to brain tumor, says, "while the masking of symptoms is a matter of not uncommon experience, the mimicry of tumor by hydrocephalic conditions is not unusual." Case XV illustrates this point. De Schweinitz and Holloway¹⁶ call attention to the frequency of scotomas in lesions of the pituitary body. Scotoma was not present, so far as known, in the two cases affecting the pituitary of this series. Cushing and Heuer¹⁷ have written interestingly of the vision field in brain tumor conditions, and state that, out of 125 cases in which perimetric observations were made, 53

¹⁴ WEISENBERG: Same reference as 2.

¹⁵ FRAZIER: Same reference as 1.

¹⁶ DE SCHWEINITZ, G. E., and HOLLOWAY, G. B.: "A Clinical Communication on Certain Visual-field Defects in Hypophysis Disease, with Special Reference to Scotomas," *Journal of the American Medical Association*, September 21, 1912, vol. 59, p. 1041.

¹⁷ CUSHING, HARVEY, and HEUER, G. J.: "Distortions of the Visual Fields in Cases of Brain Tumor," *Journal of the American Medical Association*, July 15, 1911, vol. 57, p. 200.

showed simple color interlacing or inversion, with more or less constriction of the field boundaries, and in ten of these dyschromatopsia either actually preceded any recognizable ophthalmoscopic change in the eye-grounds or accompanied incipient stages of choked disk. While several of the cases reported showed disturbance of the visual field, but few perimetric observations were made. This was chiefly due to the fact that accurate charts could not be made because of the mental confusion of the patients.

The study of the cases presented showed that the blood-pressure was not increased because of increased intracranial pressure in a single instance. The highest blood-pressure noted was 150 systolic, and this in a man fifty-six years of age (Case XIV).

Lumbar puncture was performed on all of these cases, and on some of them several times, and the chief effect noted was lessening in the severity of the symptoms. The spinal fluid pressure was thought to be increased in each instance. The globulin reaction was increased without evidence of syphilis in Cases I and VI, and there was an increased cell count (22) in Case VI.

The apparent rapidity of the growth of these tumors is striking. There was, on the average, about four months between the first symptoms complained of and the time the patient was brought for diagnosis, and about five months from the first symptoms to the time of operation, and those who died, with the exception of Case III, died shortly after operation. In one case (XV) there was only three weeks between the first symptoms and diagnosis, and a month between the first symptoms and death.

The instance of injury is so striking at times that, to the author's mind, there must be some relation between brain trauma and brain tumor in certain cases. We know that a relationship frequently exists between injury and carcinoma of the breast and elsewhere, and also, at times, between injury to bone and sarcoma. The relationship between brain trauma and brain tumor was carefully studied in each of these cases. In Cases XIII and XV the first symptom almost immediately followed a head injury, and both of these were children. In Case XII, another child, there was a head blow one year before symptoms. In Case III there was an injury two years previous, and in Case XI there was an injury, after which the patient was unconscious for thirty minutes, four years previous to the first symptoms.

In Case VI there was a severe remote head injury. It is interesting to note that in each instance in which, when an injury was closely followed by a brain tumor, as in Cases III, XI, XII, XIII, and XV, the growth was found to be a glioma.

There is a definite relationship at times between brain tumor and infectious disease. Case I seemed to follow appendicitis, Case IV had syphilis, Case VII carcinoma, Case X was thought to follow whooping-cough, and Case XIV some unknown infection. Only in Cases V, VIII, and IX was there no instance of injury or disease.

The question of the value of radiography in brain tumor diagnosis is answered, in the study of these cases, by the statement that it proved valuable in Cases I and II, showing a deformed sella turcica; in Case V by showing a calcified area in the tumor substance, and in Case XV by showing a thinning of the skull, especially in the occipital region, which was thought and proved to be indicative of tumor. The only other case in which an X-ray was taken was Case XIV, and these plates were negative. It seems significant that in four out of five selected cases for radiography the X-ray was helpful to diagnosis.

Lumbar puncture, ventricular puncture, or decompression will give relief from headache. Headache was present in thirteen cases and absent in two. It had localizing value in Cases I, III, IX, and X, and was at the opposite pole of the brain in Case XII.

Vomiting was present in ten cases, absent in five.

Vertigo was present in nine cases, absent in five, and not recorded in one.

Choked disk or optic neuritis was present in eight cases, absent in six cases, and doubtful in one (Case VII). In most of the eight cases in which it was present it was a very late manifestation. Exophthalmos was present in only one case of this series.

In Cases VIII and XIV there was no headache, vomiting, or vertigo, and slight blurring of the disks was a late symptom. In Case II there was no vomiting, vertigo, choked disk, or neuritis—only general headache.

In Cases I and VIII both hemispheres were invaded by the same tumor, and in Case VII the tumors were multiple and in both hemispheres, while in Case IV there was a tumor in one hemisphere and a hemorrhage in the other.

An elevation of temperature may be confusing in brain tumor and make one think of abscess, as in Case XIV. When it occurs it may be evidence of infection elsewhere.

The large size of some of these tumors is noteworthy, as Cases I, III, and VIII.

In cases of brain tumor involving the motor area, as in Cases IV, VIII, and XI, the mode of involvement was a gradual extension from one centre to another, and not the more or less rapid diffused hemiplegia seen in cerebral hemorrhage or thrombus.

Another point of interest is that syphilis may exist and still have no known relation to a brain tumor, as in Case IV.

In each of these cases the ante-mortem diagnosis of brain tumor was made, except in Case XIV, in which the diagnosis was made of either a tumor or an abscess. The location was correctly diagnosed before operation in Cases I, II, III, V, VI, VIII, IX, X, XI, XII, XIII, and XV, with two modifications—in Case V the fact that the tumor was pineal in origin was not thought of and in Case XV the tumor was thought to be on the inferior surface of the cerebellum and was found to be on the superior surface.

In Case IV the tumor was found to be in the opposite hemisphere, but there was a hemorrhage where the tumor was thought to be. In Case VII the multiple growths were thought to be a single frontal tumor, and in Case XIV a frontal growth was thought to be temporo-phenoidal.

The question—Is operation justifiable?—may be answered in these cases by the fact that Case XI made a complete recovery; Case II might have recovered had she not committed suicide; Case X, although much disabled, has lived six years in comparative comfort; Case III lived a year or more in comfort after an operation.

In some gliomas there may be a more or less distinct line of demarcation, although in these tumors the brain substance degenerates and the degeneration spreads by continuity. It may be that in the future cauterization as well as excision of the affected area will be considered justifiable in the operative treatment of gliomas. The use of radium is advocated by some.

An interesting theory has been advanced, that perverted ductless gland secretion may have a stimulating effect on the growth of neoplasms, and, on the other hand, that endocrine therapy in specific

instances may have an inhibitory effect on these growths. At present, however, this seems visionary.

From the study of these cases we are led to the following conclusions:

1. Brain tumors may resemble various organic and functional conditions.

2. Brain tumors do not of themselves affect the blood-pressure.

3. Lumbar puncture, when carefully performed, is not a dangerous procedure in brain tumor cases.

4. Rapidity in the development of symptoms does not mitigate against the diagnosis of brain tumor.

5. There is at times an apparent relation between head injury or infectious disease and the development of brain tumor.

6. Radiography is of value, in some cases, in brain tumor diagnosis and localization.

7. Any or all of the so-called cardinal symptoms—headache, vomiting, vertigo, and optic neuritis—may be absent in brain tumors.

8. A brain tumor may invade the tissue of the opposite hemisphere.

9. Brain tumors may attain great size without many manifestations of increased intracranial pressure.

10. The finding of a positive Wassermann reaction does not necessarily mean that a tumor is gummatous.

11. The globulin content in the cerebrospinal fluid usually is not increased in the presence of brain tumor, but may be.

12. Operative intervention directed toward the removal of the growth is justifiable in most cases of brain tumor and offers the only hope of permanent relief.

13. It is probable that, when a tumor of the brain follows an injury, pathologically it will be found to be a glioma.

A CLINICAL STUDY OF THREE CASES OF PRIMARY TUMOR OF THE HEART

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In this study only primary tumors of the heart will be considered; the secondary growths of that viscus, being of lesser interest, will not be referred to. I have been able to collect 55 cases from the literature which are clearly authentic, but I have intentionally omitted the cases of sarcoma reported by Hoffenroth, Birsch-Hirschfeld, Weiss, and Azzurini, and two cases of fibromyxoma attributed to Jägers and to Reitmann, because the original articles were not obtainable. Likewise, the doubtful cases reported by Da Costa, Hermann, Ingram, Roberts, and Bramwell are not included in the 55 cases.

In the middle of the last century Virchow described the first case of primary myoma of the heart, and he unconditionally admitted that there was with it a secondary cancer of the organ. A little later Abbers recorded a case of lipoma of the heart, Luschka a fibroma, Bodenheimer a sarcoma, and Lorne a myxoma.

A further historical notice of the question would carry the intended limits of this paper too far, as the publications on this subject are very numerous.

The presence of these neoplasms during life is usually unrecognized, and little has been written upon the clinical side of the question, for this reason. Some cases of myxoma of the heart have given rise to much discussion, as some pathologists have looked upon them as an organized thrombus, while others have considered them authentic tumors. From this discussion I shall take the opportunity of making a study of the differential diagnosis of the two processes.

From the clinical standpoint, cardiac tumors have been badly studied, their extreme infrequency being the excuse for this, so that they are rarely thought of by the physician. Although no clinical diagnosis has been made, there nevertheless exists a symptomatology belonging to them, and, although it can not be said to be

pathognomonic, it is none the less quite special and important. The symptomatology varies according to the size and location of the growth, and I shall endeavor to throw some light on this obscure question, but before this I will record the three case reports of my own.

CASE I.—Female, aged fifty-four years; housewife.

Clinical Examination.—Former health good. For 18 months, so the husband says, the patient has become irritable. She has become weak and emaciated. On March 24, 1903, after the noon meal, she was seized by a numbness in the right arm which soon disappeared.

On March 26, in the afternoon, feeling as well as usual, she walked out on some errands, when she suddenly became unconscious and fell in the street. She had convulsive movements of all the limbs and did not reply to questions. She was ordered to the hospital by her physician with a diagnosis of epilepsy.

On March 27, on her admission to the hospital, the patient was comatose. Respiration stertorous. Conjugate deviation of the head and eyes to left side. Paralysis of face, arm, and leg on right, and the right patellar reflex was absent. No contracture, nor epileptoid trepidation, but the Babinski reflex was very distinct. The pulse was tense, unequal, and irregular at 74. Cardiac dulness appeared normal in extent, and the apex beat was in the fifth intercostal space in the mammillary line. On palpation a marked systolic vibration was felt. Auscultation of the heart was difficult on account of the vesicular sounds, which masked the sounds of the heart. During her calm moments, a slight systolic murmur could be detected. Cardiac contractions followed each other at irregular intervals. No œdema. Apyrexia. Examination of the other viscera showed nothing in particular.

May 28: Condition was worse. Respiration irregular, the pulse rapid (96) and irregular, the beats being greatly disordered. Death took place in the evening without rise of temperature.

Clinical Diagnosis.—Right-sided hemiplegia from left-sided cerebral hemorrhage. Coma.

Autopsy.—The dura mater was thickened. The left Sylvian artery contained a non-adherent elastic clot. On section the large nuclei on the left side were softer than on the right.

Bronchopneumonia in early stages in each lung.

The stomach near the cardiac end presented a round ulceration with regular, abrupt edges, about the size of a quarter of a dollar. The spleen, liver, suprarenal capsules, kidneys, etc., showed nothing of note.

On section the cardiac muscle on the right was normal, so far as thickness was concerned; it was slightly diminished on the left, and its consistency was somewhat less than normal. All the cavities were dilated. There was nothing to note in the valves and aorta. Botal's foramen was occluded. In the *left auricle, on its posterior wall*, there was a polyp-formed tumor, the size of a nut, with an irregular surface, as if disintegration had taken place. It was soft and transparent. Over its surface were scattered small excrescences of softer consistency than those of the tumor and slightly furrowed.

Anatomical Diagnosis.—Tumor of left auricle, with superficial thrombosis. Commencing softening of the greater part of the left cerebral hemisphere. Commencing bronchopneumonia in both lungs. Slight bilateral hydrothorax. Gastric ulcer.

Macroscopical Examination.—The tumor was located on the posterior wall of the left auricle, near the interauricular septum. It projected upon the surface and measured $25 \times 15 \times 5$ mm. Its largest diameter was parallel to the base of the heart. Its surface was in general smooth, but on the left side there were some poly-poid projections. In color it was a light reddish brown. It was implanted by a rather broad pedicle on the endocardium. Its right edge was in direct contact with the posterior border of Botal's foramen, which was completely closed. Its base of insertion was parallel with the free margin of the foramen ovale and intruded a little upon its membrane.

Microscopical Examination.—In the fresh state examination showed that the tumor was composed of a very transparent fundamental substance which became cloudy upon the addition of acetic acid. In this fundamental substance round or stellate shaped cells were seen which anastomosed among themselves by means of prolongations.

A fragment of the growth, after having been hardened in alcohol and sections made from it, stained with van Gieson and Weigert's solutions, and also with mucicarmin-hæmalum, gave the following results: In the sections treated by van Gieson's method three very

distinct different zones were distinguished. The first was situated at the periphery, was red and represented the endocardium. Very distinct bundles of fibrillæ were seen, but were not separated by minute spaces, as in the case of oedematous tissue. In this zone neo-formed capillaries and elongated cells with a very large nucleus were seen. These cells had the character of large connective-tissue cells.

In the second zone, situated immediately beneath the one just described, the tissue was rich in young capillaries and presented a large number of hemorrhages. Some of the latter were coagulated, and fibrin was seen between the red blood-corpuscles. This second zone was composed of a finely granular or fibrillar homogeneous fundamental substance, stained a light yellow. It contained some capillaries, leucocytes, and fusiform or stellate cells around which the fundamental substratum receded slightly. The sections stained with mucicarmin-hæmalum showed that the fundamental substance took a light red-violet hue, and in the sections stained by this method a rather large vein could be seen, its lumen obstructed by a white thrombus. Some cells scattered through the preparation contained a little hæmosiderin.

In the third and most central zone the make-up consisted of fibrin, in which numerous polynuclear leucocytes and a few red blood-corpuscles were seen.

In the sections stained with Weigert's solution a large number of thin, elastic fibres appeared on the surface of the tumor. These fibres diminished in number in the areas rich in connective-tissue cells, but they were to be found throughout the preparation and even in the soft tissue. These elastic fibres were placed in all directions of the fundamental substance; they were thin and occasionally spiral. They were isolated or grouped in fasciculi.

A section made through the small excrescences on the surface of the tumor showed that they were composed of fibrin, and in their centre they contained a little connective tissue. Scattered about in the principal mass of the tissue small hemorrhages were to be seen.

This detailed microscopic description consequently showed that the tumor was composed of very soft connective tissue, approaching myxomatous tissue; that it was everywhere cut up by small elastic

fibres and contained blood-vessels and capillaries about to bud and having given rise to hemorrhage.

According to Albrecht, we may, if we take into account the composing elements, characterize this neoformation as a *hemorrhagic fibromyxælastoma*. When referring to the differential diagnosis between this type of growth and an organized thrombus I shall point out why it is impossible to admit the latter eventuality.

CASE II.—Female, fifty years old; peasant.

Clinical Observation.—The patient had always enjoyed good health, but was subject to attacks of tonsillitis. Five years ago typhoid fever was followed by perfect recovery. No cough, no expectoration. Never had acute rheumatic fever. For the past eight years she complained of moderate pains without fever, occurring every two months, seated in the region of the liver, but there had never been any icterus nor discolored stools. The patient's brother, who gave these data, said that the complaint began about two weeks previously with severe lassitude and persistent headache. The patient took to her bed; she was not feverish, but became delirious. She urinated with difficulty, and was constipated. No vomiting nor digestive disturbances. She entered the hospital January 5, 1909. with a diagnosis of meningitis or typhoid.

On entering she was delirious and restless and did not reply to questions. Temperature 100.4° F. No deviation of the face, strabismus, or paralysis of the ocular muscles. No paralysis of the limbs; Kernig's sign absent. No stiffness of the neck, Babinaki absent, no clonus. By abdominal palpation a resistance could be felt between the liver and umbilicus, which appeared to be painful. No rose spots. Spleen not enlarged. Bladder full and two litres of urine were withdrawn by catheter, containing 50 centigrammes of albumin, but no sugar or biliary pigment. Slight bilateral bronchitis. No cardiac murmurs, cardiac area of dulness normal in extent, apex perceived with difficulty. Pulse weak and rapid.

January 6: The delirium persisted. Slight Cheyne-Stokes respiration. Lumbar puncture gave issue to a clear fluid under pressure (guaiacum = 0 after centrifugal treatment). The deposit, examined microscopically, showed an average of two to three lymphocytes per field and very few polynuclears.

January 7: In the morning, as the nurse was giving the patient

some milk, a right-sided deviation of the face suddenly occurred. All the limbs became limp and inert. All the reflexes became abolished, even the ocular. Patient insensible. Pulse weak at 110. Death occurred very soon thereafter.

Clinical Diagnosis.—Tuberculous meningitis; cerebral hemorrhage?

Autopsy.—Woman of medium height. Fat well developed. No œdema of the lower limbs. Cadaveric rigidity.

The dura mater was very tense. Some clots in the longitudinal sinus. The internal surface of the dura was dry and smooth. The convolutions were flattened. On the convexity of the brain there was a little cloudy fluid, with some whitish granulations in the pia mater. The corpus callosum protruded extensively. At the base there was much gelatinous, purulent, and fibrinous exudate, and this extended into the sylvian fossæ. The arachnoid was thickened.

No liquid in the pleural cavities. A few small adhesions at the apex of the left lung. The lungs were not retracted and were somewhat full and crepitant. On section there was much hyperæmia and a little œdema.

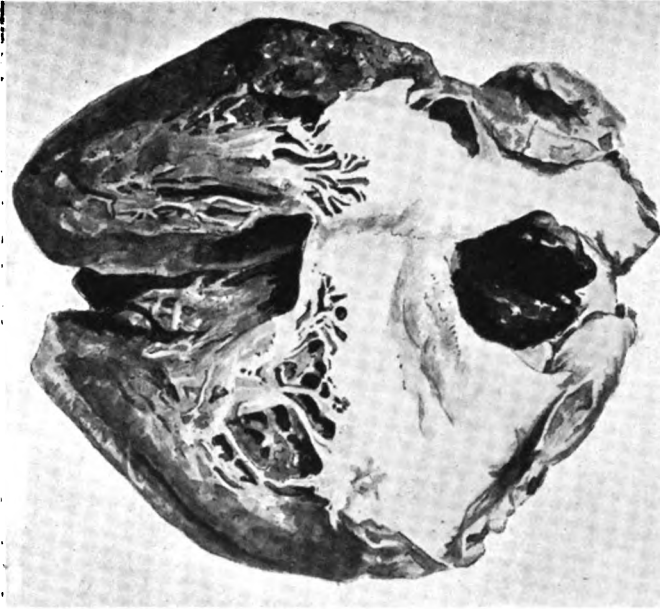
Pericardium normal and rather rich in fat. The heart was larger than the fist of the cadaver and firm in consistency. On section the muscle was reddish brown. The orifices showed nothing in particular. The aortic valves and those of the pulmonary artery were slightly calcified. The left ventricle was a little dilated, as well as the other cavities. *In the left auricle a tumor the size of a large walnut was found*, its surface fairly smooth, of a reddish-brown color. The left valve of the mitral was very large, presenting a fissure, and gave the impression of a tricuspid valve. The second mitral valve presented on its external half a small reddish-brown spot measuring 10 millimetres in diameter.

The kidneys were hyperæmic; the spleen soft and friable. The other viscera were healthy.

Anatomical Diagnosis.—Tuberculous meningitis. Tumor of the left auricle. Hyperæmia of the lungs and kidneys. Tumefaction of the spleen.

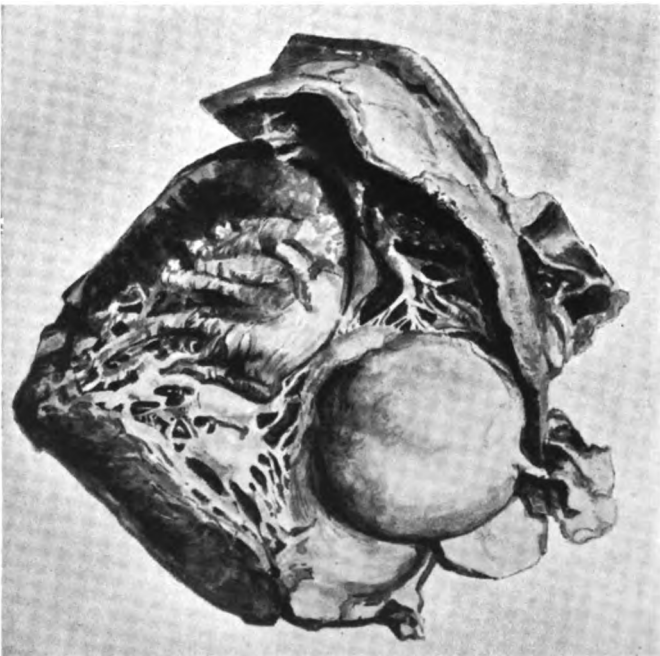
Macroscopic Description of the Tumor.—The tumor of the left auricle had a smooth surface with the exception of two small projections the size and shape of a pea. It measured $26 \times 31 \times 15$

FIG. 1



Case II. Fibromyxosarcoma of the left auricle.

FIG. 2



Case III. Primary sarcoma (spindle-cell) of the auricle.

millimetres. It was a reddish-brown color, with whitish-colored islands scattered over it; its consistency was firm. Its base of implantation, situated 15 millimetres from the insertion of the large aortic valve, exactly corresponded to the area of the formerly existing Botal's foramen, which was completely occluded and extended beyond its limits to the right and downward (see Fig. 1).

On section the color of the growth was reddish brown, and some whitish striæ were to be seen which gave to the feel the sensation of calcification.

Microscopic Examination.—The same technic and stains were used as in Case I. In the preparations prepared according to van Gieson's method it was seen that the tumor possessed a loose connective tissue which was more or less dissociated or in the form of massive fasciculi stained red. This connective tissue contained capillary vessels, and there was evidence of numerous recent and old hemorrhages, characterized by cells containing a large mass of hæmosiderin, and this pigmentation was everywhere to be seen in the preparation, both in the isolated cells and in cell clusters. There were also masses of lymphocytes here and there. With the mucicarmin-hæmalum stain the sections took a rose-violet tint. The connective tissue contained long and fusiform cells, likewise round cells with refringent protoplasm. At some spots stellate cells were to be seen. Between these cells existed a homogeneous or finely striated fundamental substance which was stained a reddish blue, while in the fundamental substance vacuolæ containing several lymphocytes, recalling the structure of myxomatous tissue, were observed. It is also to be remarked that there were clusters of nuclei surrounded by protoplasm which might have led to the assumption that there were giant-cells or a vascular bud in its formation. With van Gieson's method are already perceived bandlets, stained dark blue, situated in the midst of the fibrous tissue. These bandlets were evolved from a change of the connective tissue. In other preparations stained with silver nitrate they took brownish tint after a certain length of time. Therefore it was evident that there were spots of calcification.

Other bandlets, having an analogous structure, did not take the hæmatoxylin stain and present a refringent aspect. By staining with ammonium sulphide, even by the naked eye one could see these bandlets stained a blackish violet, and by the microscope they ap-

peared the same as the hæmosiderin, a blackish hue. Some small islands stained an orange tint could be detected by this method of staining, but no hæmatoidin crystals were to be seen.

With the Weigert method very numerous elastic fibres could be discovered in spots in the superficial layer of the tumor. These fibres, as well as partially occluded capillaries, were also visible throughout the growth, but in lesser numbers.

From this description it was evident that this tumor had a structure identical with the preceding one, and from its histologic structure it could be diagnosed as a *hemorrhagic fibromyxælastoma*.

CASE III.—Female, fifty-five years of age; servant.

Clinical Observation.—Robust, excellent health, always worked with alacrity, and never found her duties too hard. Never had acute rheumatic fever, dyspnœa, or œdema of the lower limbs.

One month before entering the hospital, and without actually suffering, she began to feel a rather severe and constant painful spot just below the left breast. She was not as strong as usual, and had some difficulty in respiration. There was no chill or fever, no cough or expectoration. A physician prescribed various drugs without benefit. The painful spot still persisted, and the patient experienced a certain ill-defined discomfort. She was sent to the hospital on August 25, 1912, with a diagnosis of pulmonary congestion.

When she entered, the patient appeared to be suffering and fatigued. There was no cyanosis of the face or limbs; no œdema; but there was a rather marked dyspnœa. Temperature 99.2° F. Some cough without expectoration.

Examination of the lungs showed dulness over the left base almost the size of the hand, and in this area could be heard tubal breathing with egophony and pectoriloquy. Over the right base there was a slight subdulness and a focus of fine râles. There was nothing to be found in the middle of the lung nor at the apex. Pulse regular but small (88); weak tension.

The cardiac dulness was transversely increased. The apex shock, which could not be distinctly felt, was in the fifth intercostal space, outside of the mammillary line. No thrill nor vibrations on palpation. Auscultation revealed a slight presystolic murmur at the apex, while to the inner side, a little above the apex, the systolic murmur was more accentuated.

Nothing to note in the digestive or nervous systems.

The urine contained the very slightest trace of albumin. After centrifugation the deposit was found to contain very few hyalin and clear granular casts. Guaiacum test = 0.

August 30: The diagnosis of mitral stenosis was made. The patient was distressed and still complained of the painful spot. Temperature 99°.

September 6: The dulness on the left had increased. The patient suffered from paroxysms of dyspnoea which appeared to be out of proportion to the size of the effusion. Paracentesis resulted in withdrawal of 1000 Cc. of a hemorrhagic fluid which did not hæmolyze after centrifugation. Cytologic examination showed red blood-corpuscles, endothelial and polynuclear cells. No bacteria. After the puncture there was an attack of pulmonary oedema. Temperature 101.3°.

September 8: The results of auscultation varied. The signs of mitral stenosis, which one day seemed distinct, disappeared almost completely the following day.

September 10: The patient's condition had its "ups and downs," but the continual dyspnoea fatigued her greatly. Traces of albumin in the urine.

September 12: Mucopurulent expectoration tinged with blood. No tubercle bacilli. Temperature 99.2° F.

September 14: Dyspnoea very intense, the nature of which could not be explained. The patient was obliged to sit on the side of the bed, with her legs in a hanging position. Although the dyspnoea was continual, it became more intense and unbearable by spells. There was a little localized oedema on the dorsal aspect of the feet. Temperature 99.4°.

September 15: With Marey's sphygmograph the line of ascension was only slight, while that of descent was prolonged. No arrhythmia. Pulse weak, 92. Temperature 99.3°.

September 16: Professor Bard saw the patient, whose very intense dyspnoea greatly interfered with auscultation of the heart. He was of the opinion that more likely there was a doubling of the first sound rather than a murmur. The systolic murmur had disappeared. Mitral stenosis was not distinct. The pleural collection was medium in quantity. Below the scapulum loud tubal breathing

was heard, giving the impression of the development of pulmonary consolidation. Temperature 100.4°.

Taking into consideration the rapid emaciation, progressive weakness, the loud tubal breathing, the hemorrhagic nature of the pleural collection, and severe dyspnoea, which could not be explained by either the cardiac lesion (the indistinctness of the mitral stenosis) or by the quantity of pleural fluid, one may very logically have suspected a pulmonary neoplasm with pleurisy.

September 18: Blood examination.—Red blood-corpuscles, 2,728,000; leucocytes, 13,175; hæmoglobin (Tallqvist), 70 per cent. Cover-slip preparation stained with Leischmann's reagent.—Polynuclears, 87 per cent.; mononuclears, 12 per cent.; transitory forms, 1 per cent.; eosinophiles, 0.

September 21: The general condition continued to decline. The dyspnoea was so severe and the discomfort so great that the patient wanted to die. The œdema over the malleoli became very marked. Temperature 99.3°. Pulse weak, rapid but regular (110–120). Auscultation of heart impossible. Urine scanty; average daily amount, 600 grammes, containing a trace of albumin.

September 23: Patient got up to urinate and died shortly thereafter.

Clinical Diagnosis.—Mitral stenosis. Left-sided pleurisy. Cancer of the lung (?).

Autopsy.—Woman of small build in complete cadaveric rigidity. Slight malleolar œdema. No decubitus. Emaciation. Skin pale.

Weight of brain, 1245 grammes. Meninges soft, injected, and œdematous, but easily decorticated. Cerebral substance very firm and humid, without anything further of note. Hyperæmia of the gray matter.

No liquid in the peritoneal cavity. Intestinal loops distended, grayish, and humid. Great omentum contracted. The liver extended four fingers' breadth below the false ribs. On the right the diaphragm reached the fifth rib. On the left it projected under the hypochondrium and extended beyond the costal border to the extent of one finger's breadth. The spleen was pushed down by it.

The left pleural cavity contained 2000 Cc. of yellow fluid bordering on green, purulent and containing fibrous masses in suspen-

sion. The lung was pushed upward and flattened against the mediastinum. The right lung was adherent.

The pericardium contained about 100 Cc. of liquid of the same nature as that of the pleura. Both layers were covered by a layer of fibrin, but it was not a villous heart nor even the beginning of that process.

On incising the heart *in situ* an abnormal resistance could be detected in the left auricle. Upon palpation a rounded mass of marked consistency could be felt, projecting through the auriculoventricular orifice into the left ventricle. A complete exposure of the thoracic viscera was then made, and it was then seen that the mass was localized in the heart and did not extend into the vessels.

The heart was then separated and presented the following phenomena upon section: In the left auricle was a tumor the size of a hen's egg. The growth had a smooth surface and was nowhere adherent. Its starting-point was in the auricle, filling the cavity almost completely, and projected into the ventricle by passing through the auriculoventricular orifice, which was virtually obstructed. However, a probe introduced by way of the ventricle could be made to pass around the tumor so that the passage of the blood was not completely obstructed. The apex of the heart was rounded.

The right auricle was dilated and its muscle hypertrophied. The fleshy trabeculae were distinctly projecting. The endocardium of the right auricle was thickened and whitish in spots. Botal's foramen was closed. The fossa ovale of the right auricle appeared rather small. The auriculoventricular orifice was enlarged, and the tricuspid valve presented a few whitish lines. The free border of the tricuspid was rounded in some places. These changes correspond to those ordinarily met with in relative insufficiency.

The right ventricle was distinctly hypertrophied, its muscle measuring from 3 to 4 mm. The fleshy trabeculae, as well as the papillary muscles, were firm and hypertrophied. The subpericardial fat somewhat penetrated the muscle. No changes in the pulmonary valves.

The hypertrophy of the left auricle was greater than that of the right, its muscle measuring 2 mm. in thickness in some places, and this hypertrophy extended somewhat on the walls of the pulmonary veins. After these were opened the dilatation of the left auricle could be distinctly seen, with a thickened white endocardium, slightly

reticulated on its surface. The mitral valves were also whitish and thickened. Circumference of the aorta, 65 mm. The aortic valves were normal. A few small fatty spots on the aorta. The hypertrophy of the left ventricle was not very pronounced; its muscle, cut obliquely, measured 14 mm.

The left lung was diminished in size. On section the parenchyma was dry; no œdema. Moderate hyperæmia of the lower lobe. Marked brown induration. Severe hyperæmia of the bronchial tubes. Right lung increased in size, adherent, and offered the same changes as in the left viscus.

The capsule of the spleen was somewhat thickened. On section the characteristics of an infectious spleen were discerned.

The kidneys decorticated easily. Surface smooth, hyperæmic. Parenchyma cloudy.

The liver weighed 1675 grammes. Surface red violet. Same color on section. The lobules were very indistinct.

Uterus enlarged, with numerous myomatous nodules of various shapes and sizes. Nothing of note in the other viscera.

Anatomic Diagnosis.—Tumor of left auricle obstructing the auriculoventricular orifice. Left purulent pleurisy (encapsulated diplococcus in cover-slip preparation) with collection. Purulent pericarditis with collection. Brown induration of both lungs. Bronchitis. Obliteration of right pleural cavity. Infectious spleen. Multiple uterine myomata.

Macroscopic Description of the Tumor.—The growth started from the interauricular wall and extended to the large mitral valve, upon which it rested. The valve was compressed and pushed downward. The portion of the tumor which freely projected into the left auricle measured $3 \times 5 \times 3$ Cm. It was spherical in shape. With the exception of a few small nodules, its surface was smooth, bright, and glistening. Its posterior aspect was covered by vessels gorged with blood. It was very hard. By pushing the fossa ovale with a finger introduced into the right auricle, which could not be distinguished by its thinness or its transparency from the rest of the endocardium, one could readily conclude that the tumor did not arise from it. The mass which projected into the auricle was only a portion of the tumor, which, after a strangulated part, continued into the auricle by a pedicle, and it was here that its starting-point was to be found. Ex-

cepting at its point of implantation, it was free and adhered to no spot (see Fig. 2).

A longitudinal section made in the auricle showed that the latter was filled by the tumor, excepting at its free extremity, where a cavity existed in which a small red thrombus, 7 mm. long, was found. On the posterior aspect of the endocardium, upon which rested the flattened part of the growth, a few small brownish spots were visible.

Microscopic Description of the Tumor.—The same technic was employed as in the first two cases. A fragment of the pedicle situated in the auricle and two pieces removed from the free portion projected into the auricle. The sections showed the same picture, so that a general description will be given. However, it is proper to remark that in the preparations made from the first fragment the auricular endocardium was thickened at the point of implantation of the tumor, and, while the thickened endocardium was fibrous and sclerous, the tissue of the neoplasm had an entirely different structure.

In sections stained with van Gieson's method, all that could be seen was a field of elongated cells with fusiform nuclei and rounded ends. The cell protoplasm did not stain yellow; it was rather more of a light rose color, and appeared to end in an intermingling with the fundamental fibrillar substance. These cells, grouped in bundles, typically represented the structure of a spindle-cell sarcoma. The fundamental substance, very little developed in certain parts, was elsewhere more abundant and homogeneous. The neoplastic tissue was everywhere invaded by blood-vessels, although scantily, and by trabeculæ which possessed a special interest.

These trabeculæ were stained red at their periphery and yellow in the centre, or entirely in red. They were different in size, averaging about that of a vertebral bone trabecula. In other sections, where the staining with hæmatoxylin was brighter and the yellow stain weaker, one could see that the entire trabecula, or its centre, had taken a dark-blue tint, as if it were a hyalin or osteoid trabecula having a calcified centre.

It was therefore necessary to ascertain if this was a hyalin connective tissue having undergone central calcification or an osteoid tissue presenting foci of ossification. By van Gieson's reagent isolated cells were found at the periphery of these trabeculæ, and in sections submitted to decalcification one saw in spots, in the interior

of the trabeculae, lacunae, each filled by a cell. Therefore, if there were no typical osseous trabeculae, there were at least some which, from the presence of lacunae filled with cells, recalled absolutely osseous trabeculae developed by metaplasia from the fundamental substance.

The sections stained by Weigert's method showed that there was no elastic tissue in the tumor, but in the sections cut from the first fragment of tissue numerous elastic fibres were seen in the endocardium. Staining with mucincarmin-haemalum was negative, as might have been expected.

The differential diagnosis was, in this case, of the simplest character. It was useless to consider organized thrombus, and as to metastatic myoma, which should have been recalled since uterine myomata were found, it could be set aside, because nowhere were any characteristic unstripped fibres to be found. Therefore the case apparently was a spindle-cell sarcoma with formation of trabeculae resembling osseous trabeculae.

TUMORS VS. ORGANIZED THROMBUS

The now-old statistics of Chambers, Willigk, Uskoff, and Napp show that secondary tumors of the heart are uncommon, while primary neoplasms are still more so. Out of a total of 3000 autopsies Thorel did not find one, while I found three instances (two fibromyxelastomas and one sarcoma) out of a total of 3745 autopsies performed at the Pathological Institute of the University of Geneva during a period of seven years. As Karrenstein has remarked, these tumors are equally frequent in both sexes, and the age plays no part.

The left heart—and the left auricle in particular—is their seat of predilection. Out of a total of 40 intracavitary tumors that I have collected from the literature, 75 per cent. were in the left auricle, nine of which were near the foramen ovale, or about 33 per cent., while five were in the left ventricle (one only was in the right auricle and four in the right ventricle).

Their histologic structure is most varied. The reporters of cases have described carcinomata, sarcomata, lipomata, myomata, fibromata, fibromyxomata, myxomata, and mixed neoplasms. It is proper, however, to remark that if by cancer a malignant growth with epithelial tissue is understood, the cases published as such are not at all

conclusive. The tumor has been insufficiently described, and the microscopic study is wanting.

So far as I am aware, a teratoma of the heart, or any tumor of this organ whose origin can be attributed to a heterotopic graft, has never been demonstrated. Joël's case is a teratoma of the pulmonary artery situated within the pericardium.

The myxomata, fibromyxomata, and mixed tumors are unquestionably the most interesting primary neoplasms of the heart, not only because they are the most numerous, but because the pathologists are not in accord as to their nature. Stahr does not even hesitate to state that the majority of tumors of the endocardium are merely vulgar organized thrombi, and not true blastomata; only a few may be myxomata or hemangiomata.

In face of this divergence of opinion, it seems to me important to examine the characteristics on which one can base his argument in order to establish a differential diagnosis between true neoplasms and organized thrombi.

Microscopically one must consider: (1) *The size*. Contrary to Stahr's opinion, it appears to me difficult to admit that an organized thrombus can attain the size of an apple or a hen's egg. Everywhere else in the body these thrombi have a tendency to retract and to become almost completely effaced. (2) *The localization*. How are we to understand the great frequency of these processes in the left auricle, and especially their localization near the foramen ovale?

Brenner is of the opinion that organized thrombi are rare on the interauricular wall, but he admits that when the foramen ovale does not close it becomes, on the contrary, a point of predilection. Marchand had, unfortunately, admitted before Brenner the same reason to explain the production of neoplasms in the region of Botal's foramen, but since this developmental disturbance has been evoked in both eventualities, we can not take it into serious consideration.

However, thrombi are quite as frequent in the ventricle as in the auricle—if not more so—and it is therefore astonishing, if the process were really a thrombus, that, out of a total of 36 tumors, only five arose in the ventricle, as against 31 in the auricle.

For this reason I prefer the two following hypotheses which apply only to tumors. Ribbert believes that there may perhaps persist embryonal mucous remains in the valve of the foramen ovale, and he

proposed to search for them in the newly-born. Thorel considers the possibility of a neoplasia of the cellular tissue into typical mucous tissue; in other words, a return to the embryonal state.

Microscopically, the differential signs are not lacking.

1. Stahr pretends that myxomata are distinguished from organized thrombi by their poverty in cells. In this I agree perfectly, and I took pains to point this out in Cases I and II, when it was stated that the connective tissue was very abundant, but almost entirely deprived of fibroblasts. These are, on the contrary, very numerous in thrombotic productions undergoing organization.

2. According to the same writer, the richness in blood and the very great number of hemorrhages would be contrary to the diagnosis of myxoma. But these hemorrhages may be very well explained by the mechanical conditions to which intracavitary tumors are submitted.

Brenner distinguished two phases of pressure for these tumors; namely, a positive one, occurring during ventricular diastole, while the vessels of the growth are gorged with blood, the auricle pressing on the neoplasm, and the interauricular walls (the base of the tumor) entering into contraction. The other is a negative one during auricular diastole, while the base of the tumor is broadened and the vessels can empty themselves. For that matter, the production of hemorrhages may also be explained by the fineness of the capillaries and their great number (Karrenstein). What is still more characteristic in my first two cases is not the amount of the hemorrhage, but the presence of old hemorrhages besides recent ones.

3. The richness in the blood-pigment, which to Stahr appears to be an argument in favor of organized thrombi, does not seem to me at all convincing. All tumors, in fact, may contain hæmosiderin. On the contrary, hæmatoidin deserves more attention. In organized thrombi hæmosiderin is found at the periphery and hæmatoidin in the centre. The latter may also be preserved without changes taking place for a long time. Now, I was unable to discover hæmatoidin in the first two cases, while hæmosiderin was found throughout their structure in large quantity.

4. The presence of elastic fibres disseminated throughout the entire tumor mass is not compatible, either, with the idea of an organized thrombus. I was able to demonstrate that these elastic fibres

were to be found in considerable number at the surface of the tumor, and that they gave the impression of a thickened endocardium. They continued with the elastic fibres of the auricular endocardium, but this continuity was perhaps not sufficiently put in evidence. It showed that the endocardium covered the tumors and that, consequently, their starting-point was subendocardiac.

5. Finally, the picture of an organized thrombus is quite different from that seen in Cases I and II. The organization of a thrombus commences by the penetrating of the capillaries coming from the periphery, and the connective tissue forms after this. Now, quite on the contrary, I found a neoformation of capillary vessels in the midst of the fundamental substance in fully organized tissue.

Therefore I feel that I am authorized to say that Cases I and II certainly represent, according to their composing elements, fibromyxelastomata and not organized thrombi. At this point I might still discuss a theoretical question; namely, whether I should class these two tumors among the true blastomata or among the processes of malformation. Modern pathology endeavors to separate true progressive blastomata from hæmatomata, a kind of growth which, being the consequence of an embryonal disturbance, gives rise to a local proliferation which is rather more of a neoformation from excess.

As to Case III, the growth is malignant from the histologic viewpoint, since it presents the characteristics of a sarcoma. Should a special origin different from other growths be attributed to it on account of the areas of ossification of the trabeculæ which were described? We might, in this case, advance the idea of an embryonal graft of skeletogenous tissue, but we know that in chronic endocarditis one sometimes finds osseous or even cartilaginous tissue present. We therefore can not deny the function of the endocardium for producing osseous trabeculæ. A tissue capable of such a production in cases of inflammatory processes is still more so in the case of neoplasms.

CLINICAL CONSIDERATIONS

Bodenheimer, the first writer who essayed to give a clinical study of primary tumors of the heart, thus expresses himself:¹ "The symptomatology of cardiac tumors is still a vast field open to investigations, because of the insufficiency of detailed clinical reports. This

¹ *Inaug. Dissert.*, Bern, 1865.

is the opinion of most clinicians, . . . but this opinion is in contradiction with the results of the study of already known cases. In omitting those cases in which vast lesions of other organs veil the clinical picture, and only stopping at notoriously primary tumors of the heart, we come to distinguish certain symptoms which effectively depend upon these lesions."

This writer adds that the symptomatology of cardiac tumors offers nothing precise or well determined, and that it depends upon the size of the growth. If the tumor occupies a position which does not interfere with the heart's action, it will not set up any morbid phenomena. If, on the contrary, it occludes the cardiac orifices, phenomena of stenosis or insufficiency will arise. The only symptoms noted have been disturbances of the venous circulation. The diagnosis, in the cases where the symptoms are not marked, can be made only by exclusion. However, Bodenheimer refers to the relative ease of the diagnosis of neoplasms of the right heart in contrast to the rarity of defects of the right heart, whose symptoms in some cases may be rather more in favor of a tumor of a cardiac malformation.

Ely² insists on the fact that primary tumors of the heart do not give rise to much of any disturbance nor to any characteristic symptom. Fränkel maintains that a pericardiac hemorrhagic fluid collection recurring quickly after puncture when the patient is not in receipt of a traumatism and has neither tuberculosis nor scurvy almost surely indicates the presence of a cardiac neoplasm. Eichhorst, in his work on diagnosis, states that he does not believe in the possibility of the diagnosis. Some tumors remain latent on account of their small size or their situation. Others grow on the valves or in the immediate neighborhood of the cardiac orifice and give rise to symptoms of stenosis or insufficiency, the cause of which remains unknown during life.

Berthenson³ closes the histories of his cases by the following remarks: "In the cases where the clinical picture of the disease is indistinct and presents deviations from the normal type which can not be accounted for, multiple emboli may give rise to the idea of a cardiac tumor, particularly that of an intracavitary growth."

² *Thesis*, Paris, 1874.

³ *Arch. de méd. expérimental*, 1893.

Victor Petit⁴ has given a clinical study of myxomata of the heart. According to his way of thinking, primary cardiac tumors are of a character peculiar to themselves; namely, an intermittence of the symptoms. This will depend on the movements executed by the growth around its pedicle. One may hear a murmur, but its intensity, its note, and its seat will vary, and, what is still more important, it may alternately appear and disappear. Such are the elements by which the hypothesis of an intracavitary tumor may be upheld.

Krell believes that one may also make a diagnosis by an important development of a certain portion of the heart, as well as a marked stasis in the territory of a vein, while regions usually involved in cases of cardiac weakness remain free.

Link,⁵ in an important article, considers that it is useful to divide primary tumors of the heart into four groups, for the study of their clinical symptomatology. In the first group he places tumors developed in the auricles. The characteristic of these neoplasms is to produce a very marked stasis in the lesser as well as the greater circulation. During life it may have been impossible to explain this stasis, which did not depend upon any ordinary cause.

The second group comprises pedunculated tumors arising in the left auricle, filling its cavity and sometimes penetrating through the mitral orifice into the left ventricle. These neoplasms produce symptoms of stenosis or insufficiency with disturbances of compensation. The signs found on auscultation are frequently very variable and change according to the position of the patient. Sudden death occurring in some cases was explained by the penetrating of the tumor into the auriculoventricular orifice.

The third group is composed of tumors having as their principal seat the right ventricle. The clinical symptoms have been about the same as those of angina pectoris with sudden death.

In the fourth group Link places two very different cases, that of Bucquoy (a cancer of the left ventricle and lower wall of the right ventricle arising, in all probability, from a cancer of the ovary); second, that of Prudhomme, of a male, twenty-four years of age, who presented œdema, dyspnoea, and undoubted signs of aortic in-

⁴ *Thesis*, Paris, 1896.

⁵ *Zeitschrift für klin. Med.*, 1909.

sufficiency. Autopsy showed that, of the three aortic valves, only one was intact. The other two presented each a vegetating concretion having the form of a feather duster. The posterior valve was perforated at its base by the growth, which unfolded on the internal valve after its emersion. Bucquoy considered the tumor to be a melanæic cancerous vegetation emerging from an infravalvular ulcer of the same nature, but unfortunately a histologic examination is wanting. Both these patients died a sudden death, but had not offered any special symptom during life.

Link ends his paper by saying that one should be able to make a diagnosis, but he does not indicate by what means.

Such is the summary of the literature concerning the *clinical* aspects of primary cardiac growths. Although small, it is important, and its interest does not seem to me to deserve the disdain accorded it by text-book writers. The chapter on the semiology of the heart is the one which clinicians should chiefly consider in order to add to the conquests of modern medicine. Although I have no pretence to completely fill up this lacuna, I think that it is useful, for the clinical study, to group cardiac tumors according to their site, and I propose the following division: (1) *Valvular tumors*; (2) *intramuscular tumors*; (3) *intracavitary tumors*: (a) *sessile*, (b) *pedunculated*.

1. *Valvular Tumors*.—I have been able to collect 11 cases coming under this heading. It is unfortunate that clinical remarks are wanting in six, because we can not make use of them for this reason. The tumors had different sites: mitral valves, tricuspid valve of the pulmonary artery, free border of aortic valve; while in size they varied from that of a millet-seed to that of a bean or cherry. It would have been interesting to have known the result of the clinical examination of the heart. In one case the growth was an autopsy finding.

On the contrary, important clinical signs accompanied four of the cases. Death occurred suddenly or after a progressive cardiac cachexia. In Debove's case a tumor with a short pedicle, situated on the upper aspect of the tricuspid, was to all appearances engaged in the right auriculoventricular orifice and resulted in death of the patient in fifteen minutes from asphyxia.

In Prudhomme's case, already referred to, the cauliflower growth on the aortic valves produced undoubted signs of aortic insufficiency,

a fact to be readily accounted for, and Duroziez's double murmur was the only symptom wanting.

Progressive cardiac cachexia with symptoms of insufficiency and mitral stenosis observed in Leonhardt's case was a natural consequence, since a tumor the size of a cherry existed on the internal mitral valve.

Therefore it is evident that primary tumors of the cardiac valves remain either silent and unsuspected or else produce phenomena of stenosis or insufficiency and consequently can not be clinically distinguished from lesions produced by inflammatory processes of the valves. The diagnosis, for these reasons, seems impossible to make during life, and the most that can be done is to consider the possibility of a valvular tumor when signs of insufficiency or stenosis appear rapidly in a patient who did not present any former evidence of an official lesion or any etiologic factors common to these affections.

2. *Intramuscular Tumors*.—Primary intramuscular tumors of the heart are invariably malignant, and just so soon as they have given rise to signs clinically noticed the affection undergoes its evolution with great rapidity. Of the seven cases I have been able to collect, all the patients died within a few months, and the longest evolution noted was, at most, two years.

At the commencement the patients complain of rather intense precordial pain, great distress, dyspnoea, palpitations, and general weakness. In the period of full development a progressive oedema occurs along with a progressive cardiac cachexia. A critical examination of the patient reveals nothing, or almost nothing, which can explain the clinical manifestations.

The pulse is regular, but with a low tension, and varies much. It is usually rapid; in Drysdale's patient it was 84, in Link's it varied from 120 to 164, and in Fränkel's case it was 120 to 140. This regularity of the pulse-beat, regardless of the fact that there is a considerable destruction of the cardiac muscle, is most remarkable. One may even well ask how the heart was able to continue its functions for so long a time in Link's case (two years and six months), in which only healthy tissue one millimetre in thickness existed between the tumor and the auricular cavity.

Stokes-Adams's disease has never been observed in these cases.

The heart is usually increased in size, its dulness may extend two

to three fingers' breadth beyond its normal limits. The heart-sounds were weak, sometimes extinguished, but regular, excepting in Drysdale's case. Bodenheimer and Link mention a swelling of the veins of the neck without pulsation.

As to other lesions occurring during the evolution of the process, in three cases a pericardiac fluid collection was accompanied by a bilateral or unilateral pleurisy. In Fränkel's case the pericardiac fluid was hemorrhagic and re-formed rapidly after puncture.

Such is the symptomatology of intramuscular cardiac growths. Let us now see if it offers anything very characteristic. It certainly shows that the affection is seated in the heart, since the patients present certain signs of cardiac weakness and marked stasis. But an exact diagnosis has, as yet, never been made, so far as I am aware. Biermer was nearest to the truth when he said: "Heart-disease of unrecognized cause." In fact, absence of signs on auscultation eliminated valvular trouble; heart-sounds were regular, although rapid, did not call attention to the muscle itself or to a disturbance of the innervation. Therefore there was some heart trouble of unrecognized cause.

It is just the want of concordance between the clinical signs and the morbid symptoms which should have commanded attention, and this is evident in all the reported cases.

I therefore think that, when a subject presents a clinically inexplicable progressive cardiac cachexia, the physician would be right in suspecting a primary intramuscular cardiac neoplasm. Fränkel's sign, considered as almost pathognomonic by some writers, appears to me less important than the clinical discordance just alluded to.

3. *Intracavitary Tumors.*—These growths are the most interesting, not simply because they are the most numerous, but because they have given rise to close study.

Of the forty cases that I have collected of very different sites of the growth, in four instances it was the right ventricle, once the right auricle, five times the left ventricle, and thirty times the left auricle. It appears to me useful, for the consideration of the subject, to divide the tumors of the right heart and those of the left heart into two subdivisions, according to whether they are sessile or pedunculated.

RIGHT HEART.—Of the five cases, there is no clinical history in three, on account of the position or small size of the tumor. The other two offered important clinical signs.

(a) **Sessile Tumors:** Hornowski's case was a tumor which, in developing in the right ventricle, had suppressed the function of the tricuspid valve by pushing it from below upward. Clinically, there was a very intense systolic murmur over the tricuspid area. The second sound was not perceived, hidden, according to the opinion of the reporter, by pulmonary tissue. The other areas of auscultation revealed nothing of note. The patient was admitted to the hospital in a dying condition, and no data as to the history, etc., could be obtained. If auscultation of the lungs had been negative, the absence of a chronic lung disease, as well as cardiac trouble—in a word, the absence of all the usual factors of a functional tricuspid insufficiency—should have eliminated this diagnosis, and the only diagnosis that would remain would be that of foetal endocarditis.

(b) **Pedunculated Tumors:** A single example is to be found in this subdivision, and is a case of pedunculated lipoma, reported by Adam Brewis, seated in the right ventricle of a female child, seven years old. The growth, by obstructing the auriculoventricular orifice, caused sudden death and necessitated a forensic examination.

Tumors of the right heart are evidently quite as rare as non-functional affections of the valves. When they are seated at the apex of the ventricle they do not give rise to any clinical sign so long as their size is not sufficient to cause an obstacle to the circulation. When seated near the valves they produce stenosis or insufficiency, with the entire train of symptoms common to them. In the auricle, when their development is sufficient, they cause a dyspnoea of exceptional intensity, with very marked hindrance of the right venous circulation. Death occurs easily and suddenly from obstruction of the right auriculoventricular orifice, or slowly after phenomena of stasis and cardiac insufficiency have occurred.

The diagnosis can be made only by exclusion. A tumor should be suspected when a subject presents phenomena of compression of the right auricle or a non-functional right cardiac affection to which no satisfactory clinical explanation can be attributed.

LEFT HEART.—As I have already stated, the left heart is the site of predilection for primary neoplasms (thirty cases in the auricle, five in the ventricle). Nearly all the patients offered important and very characteristic signs when the growth was pedunculated.

(a) **Sessile Tumors:** Two cases had no clinical history. All the

others offered symptoms of an organic cardiac affection—nine in all, including Case I. The patients complained of weakness, shortness of breath, and palpitations. They experienced constant oppression and occasionally the sensation of smothering; also, cyanosis of the lips and a slight malleolar œdema.

Auscultation gave indistinct cardiac sounds, or there were signs of mitral insufficiency with occasionally a slight degree of stenosis. Cardiac weakness increases and results in death, which may often be hastened by an intercurrent disease, or the end may come suddenly from cerebral embolus. The latter process, whose cause is still unrecognized, has been mentioned by some writers, and I shall revert to it once more further on. They are due to small thrombotic masses situated on the surface of the growth, or to particles of the tumor which become detached and enter the general circulation.

Consequently sessile tumors of the left heart can in no way be distinguished from an organic cardiac affection.

(b) *Pedunculated Tumors*: These also produce general symptoms, the usual clinical picture being a more or less marked œdema of the lower limbs, palpitations, pain in the cardiac region, dyspnœa, and fearful distress. The dyspnœa may be excessive.

In Case III the dyspnœa was such that I have never seen its equal, and at the time I was struck by its disproportion to the other clinical signs. The cardiac lesion was indistinct, even uncertain, because in the horizontal position a systolic murmur could be heard, which changed to a variable presystolic murmur in the sitting position. The pleural collection, medium in quantity, had not pushed the heart aside. No cyanosis nor any important stasis or ascites, and, at most, a very slight malleolar œdema. Only a trace of albumin in the urine.

The dyspnœa obliged the patient to bring the inspiratory muscles into play and remain seated on the edge of the bed with the legs in a hanging position. Pleural puncture gave no relief, but quite the contrary. It evidently was more of a cardiac than a pulmonary dyspnœa, but it was impossible to find a satisfactory cause for it. It was due to the tumor, as large as an egg, which filled the left auricle and obstructed the auriculoventricular orifice.

The *pulse* of these patients has not aroused any attention. Usually it is small, frequent, and often irregular. By the use of digitalis it became slower and more regular in Waldvogel's case and that of

Link, but this action soon ceased. In Robin's case and in Case III the radial tracings, taken with Marey's sphygmograph, showed a short oblique line of ascent and a long line of descent without dirotism. The pulse was weak and slow. Some writers have found that the amplitude of the pulse did not correspond with the force of the cardiac impulsion.

HEART.—The dulness was increased in almost all the cases. Auscultation usually revealed a systolic murmur of a rather intense character, sometimes a drumming sound or a presystolic murmur, but all these signs presented one peculiar character—*their great degree of variability*. This belongs to pedunculated intracavitary tumors. Thus, in Lorne's case, the systolic murmur at the apex was detected only after several days; in the case of Thompson and Atchison the systolic and presystolic murmurs became less marked and disappeared, leaving only a pure systolic murmur. Blochman found a presystolic murmur of varying intensity in his case. In Marchand's case the murmur was at times systolic, at others diastolic. In Bacmeister's case the heart-sounds were pure in the first place; then a slight systolic murmur appeared at the apex and was absent from time to time.

Five physicians auscultated my patient (Case III), and discord ensued when it was a question to determine the site and nature of the murmur. Doctor Roch, chief of clinic, and myself were of the opinion that the diagnosis was mitral stenosis. At the beginning there was a slight presystolic rolling sound at the apex and a more marked systolic murmur above and inward from the apex. Later, as the patient could no longer remain in any other posture than sitting, on account of the dyspnoea, auscultation was more difficult. Nevertheless, it was easy to detect the disappearance of the systolic murmur, and only the presystolic rolling persisted, which itself varied from day to day.

This extreme variability of the auscultation signs in subjects who are in no way neurotic is most important, because no ordinary valvular lesions exist where this occurs. Its interpretation is a delicate matter, but most interesting. Unlike Victor Petit, I do not believe that this variability is due to movements of torsion of the tumor around its pedicle. For example, in my case the pedicle of the growth was much too thick and in consistency much too firm to have

submitted to movements dependent upon systole and diastole. Its immobility must have been perfect.

I am rather more inclined to believe that the variability of the auscultation signs depends upon the position taken by the growth in relation to the mitral orifice. Thus, in Case III, the tumor reached the mitral and should, from its weight when the patient was recumbent, compress and lower the mitral valve. It thus prevented it from closing and rendered it insufficient at the same time that it produced a slight degree of stenosis. In the sitting posture the tumor became engaged in the left auriculoventricular orifice, and then only produced a stenosis. When the tumor becomes thus engaged in the valvular orifices a sudden paroxysm of cyanosis of the face may sometimes be produced, but I did not observe this in my case.

Evolution.—It is fatal in a short time, as soon as the clinical signs become evident. It depends upon the histologic structure of the neoplasm and the rapidity of its development.

Death may be slow and occur after a progressive cardiac cachexia. It is frequently hastened by some intercurrent pulmonary or pleural affection, but it may be sudden from cerebral embolus, usually localized on the left side. These emboli may be multiple. They were first mentioned by Robin in 1893. His patient recovered from his first paralysis and returned to work, but two years later another embolus resulted in death. Robin had suspected the cardiac origin of these emboli, but it was impossible for him to determine upon the cardiac condition which had given rise to them. Berthenson and V. Petit insist likewise on the value of this diagnostic sign.

Death may also suddenly occur, but by a different mechanism. Thus, in Boström's case a fibromyxoma had penetrated into an already stenosed mitral orifice and completely occluded it.

The pedunculated growths are therefore to be distinguished from organic valvular affections of the heart by producing an exceptional continued intense dyspnoea for which no satisfactory explanation can be given; by producing rather indistinct, and particularly very variable, signs of insufficiency or stenosis; by sometimes producing, when the patient changes from the recumbent to the sitting position, sudden paroxysms of cyanosis due to the penetration of the tumor into the mitral orifice; and, lastly, by the production of multiple emboli, the cause of which is still unknown.

Psychiatry

THE NEUROLOGICAL ASPECT OF MENTAL BACKWARDNESS IN CHILDREN AND ITS TREATMENT *

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THIS subject has not received the amount of attention of the neurologists commensurate with the frequency of its occurrence and the ever-increasing problems confronting the educational authorities. The result is that psychologists and other well-meaning persons have attempted to grapple with this problem from some particular standpoint. In the vast majority of instances these people lack any knowledge of the anatomy and physiology of the central nervous system, as well as the prerequisite of a critical introspection. It is no wonder, then, that under such circumstances to-day this and tomorrow the other treatment as a panacea or cure-all for every conceivable kind of mental backwardness make their appearance. I need only remind you of the tonsil and adenoid hysteria, the pineal gland feedings, etc. No one would gainsay that in some instances some good may be accomplished by such measures, but only in addition to others, as may be indicated after a critical study of each individual case. For even in so-called types of patients there are not always the same underlying etiologic factors. It seems perfectly evident that such cure-alls by pseudo-scientists would complicate this problem and thereby enhance the misfortune of the suffering child and parents. Under such circumstances it may not be out of place to permit the calm and unprejudiced voice of science to be heard.

What do we mean by mental backwardness—what is its etiology, its concomitant manifestations, and to what extent can it be ameliorated?

By a mentally or intellectually defective child I mean one who

* Read before the German Medical Society, December, 1916; Yorkville Medical Society, March 19, 1917.

is far below normal standard as accepted by well-established intelligence tests. I say far, because in testing the mentality of a child several factors must be taken into consideration; namely, the personal equation of the examiner, the surroundings of the child during an examination, and its attitude, the home environment, the intelligence of its progenitor's stock, and, lastly, whether the defect is general or limited to certain tendencies. So that a child found to be one or even two years behind its age according to a certain intelligence test I would hesitate to call defective.

The etiologic factors are generally considered pre- and post-natal. They may arise as a result of trauma, of an infectious process, or, again, as a result of an affection; namely, an inherited neuropathic or psychopathic constitution.

The most frequent traumatic influences are delayed labors or instrumental deliveries. Other forms of postnatal traumata are by no means uncommon, and are mentioned in some histories of patients suffering from convulsions. I want to call attention here to psychical trauma, such as shock and fright, which is a frequent exciting factor of a neurosis, or even a psychosis on the basis of a neuropathic or psychopathic constitution.

Of the infectious agents, the exanthemata take the first rank. Those of the cerebrospinal meningitides, poliomyelitis, pneumonia, diphtheria, of various streptococcic infections of the nasopharynx and middle ear, and, finally, of syphilis and tuberculosis, play a very important rôle in the etiology.

Both the traumatic and infectious agents produce inflammatory conditions of the meninges at the base and the convexity of the brain. If at the base, some of the cranial nerves become involved and give rise to defects of the special sense organs. Blindness will result when the optic nerves are involved, and deafness when the acoustic nerves are involved. Children surviving from a basilar meningitis are very often deaf and blind. If the convexity of the brain be involved, we may deal with marked hemiplegias or slight hemiparesis, depending entirely upon the extent of the involvement. Epileptiform convulsions usually accompany such hemiparesis. If the speech centres of the brain are encompassed in the lesion, complete or partial motor or sensory aphasia may result. Indeed, I have examined children with a previous history of meningitis with

only an alexia, who, because of their inability to comprehend printed or written language, were put down in school as morons and promptly had their tonsils and adenoids removed, without, of course, effecting any improvement. A careful study of the case early in life would have indicated that reëducation of the dormant centre was imperative and the child would have been saved humiliation and distress, as indeed proved successful in another patient of mine. In cases where the communicating foramina of the ventricles are partially or completely occluded by inflammatory exudates, an accumulation of the cerebral fluid will lead to an internal or external hydrocephalus. If the amount of fluid is sufficiently large to exert undue pressure and irritation upon the brain cortex, spastic diplegias with convulsions and a very low mentality will result. Vision in such cases is always affected on account of the pressure upon the optic tract.

Cortical and basilar hemorrhages are frequently produced by traumata, which also lead to depressions of the skull, that in turn press and irritate or even destroy cortical areas. In such focal lesions we get localized symptoms in conformity with the place involved. Together with such focal symptoms, the X-ray will be of great assistance in arriving at a diagnosis.

In a large number of my cases hereditary syphilis, alcoholism, and tuberculosis are prominent etiologic factors. It may not be amiss to discuss them a little more in detail.

In the case of syphilis I make a distinction between infection and affection. In an infection the syphilitic virus, the *Spirochæta pallida*, is inoculated. Such infection of the central nervous system will result in vascular changes, gummatous exudates in the meninges and brain substance, or, again, in the so-called parasymphilitic manifestations. Blindness, deafness, convulsions, choreiform movements, paralyses, ataxias, and even psychoses may result in correspondence with place and extent of the lesion.

By an affection I mean a state of lowered vitality as compared with the normal threshold. This is the result of the chemotactic action of the toxins upon the protoplasm of the brain-cells, a pathologic change in their normal molecular consistency. Such lowered vitality is inherited by the offspring, but not the virus that produces such change. This affection manifests itself in what is commonly known as a neuropathic or psychopathic constitution. Alcohol and

tuberculosis as well are productive of such conditions. It is an accepted fact that such hereditary taints are transmitted from generation to generation. In progenies of consanguineous marriages, in whom there are such deleterious factors, these deteriorating manifestations are brought out more promptly and certainly. Upon such a constitution psychic or physical traumata or infectious diseases will engraft some form of mental deterioration and thereby render the mind decidedly unstable if not insane.

The general physical development of the infected as well as of the affected child may be retarded or even markedly inhibited. Agenesis, aplasias of various tissues, delayed development, especially of the central nervous system, are met with in no rare instances. Dystrophies are now regarded as the result of such hypoplasias. The cells may possess a weak power of resistance and fatigue rather early in life, succumbing easily to the very onerous tasks required of them in the daily discharge of their functions, unable, as it were, to proportionally assimilate new food and replenish energy so easily lost. Of the most prominent stigmata, Hutchinson's teeth, an interstitial keratitis, radiating scars around the angle of the mouth are valuable guides to a syphilitic constitution. The Wassermann reaction may be positive in the case of an infection, but obviously negative in the case of an affection.

Alcohol, even in small quantities, frequently used, affects the protoplasm and therefore the entire system. It lessens the absorption of oxygen by the red blood-corpuscles and the exhalation of carbon dioxide. It not only diminishes the powers of resistance and therefore favors the growth of pathogenic bacteria, but also inhibits to a great extent the metabolic changes in every organ of the body, thus producing a chronic toxic condition.

The decrease or increase of mental disorders and crime in a particular locality is shown by statistics to be in direct proportion to the rise and fall of consumption of alcoholic beverages.

Since the mother has a greater share in the life and care of the child, maternal alcoholism is of far greater danger than the paternal. It was proved again and again that the earlier in her pregnancy a woman takes to drink the more certain will be the debility of her offspring. Statistics show that paternal alcoholism plays a great rôle in the etiology of the defective, so that we may assume that

paternal intemperance, if not itself due to a neurotic heredity, and especially if emphasized by disease or privation, certainly produces a marked influence upon nutrition and causes mental and physical degeneration, both in parents and offspring; in other words, is productive of a neuropathic or psychopathic constitution.

In tuberculosis we also deal with a toxic condition that ravages the tissues of the parent and produces not only a diminished resistance to infections but gives rise to imperfect bodily development of the child. Such children show, as a rule, an underdevelopment of the various tissues never reaching the norm, and thereby, also, a low threshold of their functions. One notices a subnormal body weight, a positive sign of lack of proper nutrition. The skin is spongy, pale, rather inelastic and yet not tense. The muscles are, as a rule, flabby and weak, prone to easily fatigue and become exhausted. The lymphatic glands are always swollen. These are constant signs of a scrofulous diathesis, and as these children grow older they develop various organic diseases of the viscera and bones characteristic of a faulty nutrition. Hand in hand with that goes a mental backwardness of various degrees, from mere retardation to a complete imbecility.

Finally, we come to consider the diseases of the ductless glands. The secretions of these glands contain chemical substances which profoundly influence the functions of the body. According to one hypothesis they enhance the assimilation of food and thereby influence growth, and according to another they are supposed to be germicidal and neutralizers of toxins and thus prevent the destruction of tissues. Whatever hypothesis may prove the correct one, it has been established experimentally and clinically that whenever the balance of these internal secretions is upset by a lesion in any of these glands there result diseases which give rise to definite clinical manifestations. Just as the various organs are influenced in their nutrition and therefore in their growth, so are the growth and functions of the central nervous system profoundly influenced in the same ratio. All children suffering from any defect of the ductless glands are mentally below par. It matters not whether we are dealing with hyperthyroidism, giving rise to exophthalmus, tremors, tachycardia, moist skin, and fine hair, the syndrome of Basedow's disease, or its antagonist, hypothyroidism, with the symptom-complex of cretinism,

infantile myxœdema, and Mongolian family idiocy: the stunted growth, myxœdema, sunken nose, thick lips, coarse and scanty hair, dry skin, chilly sensation, brittle nails, large and pendulous abdomen, and obstinate constipation; or, again, hyperpituitarism with the syndrome of acromegaly or that of hypo- or dys-pituitarism, with stunted growth and sexual precocity or infantilism, mental backwardness is a constant accompaniment, depending, of course, upon the degree of involvement. Since these glands act rather in unison, maintaining a sort of balance, the normal secretion of each gland depends largely upon the normal function of all others. We therefore find usually more than one gland involved. The thymus, adrenals, testes, or the ovaries, alongside the other glands, are, as a rule, suffering pathologic changes, perhaps in the nature of a compensatory function. We therefore never have distinct classical types as relating to a single gland. The particular etiology of these affections is not definitely established, but many investigators have been able to show that alcoholism, tuberculosis, and syphilis in the progenitors do play a considerable rôle in their production. I want to say right here that enough attention has not been given to the defects of the ductless glands in childhood as a possible etiological factor in the production of backward mentality. I am inclined to place the onus upon them whenever I am unable to elicit any other factor in the absence of focal symptoms that would otherwise point in another direction.

From what I have said before, it is evident that the possible amelioration of such conditions may best be accomplished by prophylactic rather than curative measures. It would be impossible to enter upon an extensive discussion of all measures in this paper, but a few general remarks may prove of value.

The education of the public in eugenics is the important phase of prophylaxis. Consanguinity in marriage should be restricted by legislation to a far greater extent than is done by any church or creed. As the production of these unfortunates is not limited to any particular class of society—for they are the results of the outcroppings of various defective and vicious tendencies in all ranks and classes, including the highest, where the black sheep of the family does not come merely by chance—the avoidance of injudicious matings utterly unfit for the propagation of healthy offspring is imperative. It

should be made compulsory for any one applying for a marriage license to undergo a thorough physical examination by trained observers, and the Wassermann reaction should be resorted to in every case.

So far as curative measures are concerned, they must be both medical and educational. Hygienic surroundings and proper, nourishing, and easily assimilable diet are the most important prerequisites, and, along with these, proper medication, as the case may indicate, will, in cases which are still amenable to medical treatment, do a great deal of good. Especially is this true in cases of hereditary syphilis and disturbances of the endocrinous glands. In the former, when the Wassermann reaction has proved positive, a judicious antisyphilitic treatment has proved fairly successful in my hands. In the latter great care must be taken to find out which of the glands is primarily responsible for the defect. We know that several glands are, as a rule, involved and perhaps rather as a compensatory activity, as nature is striving to reestablish the balance. We must also bear in mind that the active principles of these glands are not harmless, and great caution therefore must be observed in their administration, lest we defeat the very purpose we desire to attain. I would urge, then, against the so-called polyglandular therapy and against large dosage. While I have not as yet seen in my own practice or heard of any reported cases of mental defect in children having been entirely cured, we have ample evidence now that in some instances proper medication will redeem an otherwise lost wreck.

Alongside medical and hygienic care an individualizing, not specializing, teacher, one with a keen power of observation and fairly well equipped with knowledge of psychology and anatomy and physiology of the central nervous system, will materially aid in bringing order out of chaos. All these measures must be attempted early if some degree of success is to be attained, for as the child grows its brain-cells adjust themselves to their environment, and to begin late would mean to attempt the impossible.

In conclusion I would say that each case must be carefully studied as an entity by itself, for, as I have pointed out, there are different etiological causes behind the same symptom-complexes, so that we must minister to its particular needs as the case may demand. This also holds true with respect to pedagogic methods. It is useless to

demand of the teacher that he apply the lockstep-stencil method in teaching these defectives, in accordance with the prescribed grade work of the curriculum. The ability of each child in every branch of academic studies must be carefully tested, and its particular inclination in a certain direction observed and developed along these lines. Industrial training adapts itself admirably in the vast majority of defectives, for we are dealing here with the concrete and simple. We must remember that we are dealing with the primitive mind, and the fewer brain areas we invoke for any given idea the sooner we shall accomplish fair results. The fewer the brain-centres involved at one time, and the more persistent the impressions become, the better their retentive memory. By catering to their particular inclination we arouse a more acute attention. And their interest can be aroused only along such lines, and their crude imagination enhanced. Above all, the educator must bear in mind that all we want is a self-sustaining individual and not an expert accountant or other professional of a higher type.

Public Health

SOME FOOD FACTS FOR WAR-TIME CONSIDERATION

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IN the preface to his book, "Treatment of Diabetes Mellitus," Joslin says: "Tuberculosis gave consumptives, and the rest of us, fresh air. The modern treatment of diabetes is giving the community very definite ideas about diet and the desirability of maintaining the body in the best possible physical condition. Between the consumptive and the diabetic we have very little excuse to be ill." This is undoubtedly true, but the aggregate knowledge of food values is still very meagre; and what the consumptive and the diabetic have failed to do will be accomplished by contingencies incident to the ravages of war—for in this case, as truly as in any other, will necessity be the mother of invention.

It is in anticipation of such a contingency that the writer of this brief article is presenting the accompanying tables, one of which (Table I) has already been published for popular reading in the *Philadelphia North American*, and, as a matter of permanent record, is herewith presented.

Accompanying each article of diet are two lines of figures. The first figure on the top line represents the price per pound of that commodity April 15, 1917, unless otherwise noted. Each subsequent figure in the same line represents a presumed advance in price. Each figure in the second line represents the fuel value in calories received for 5 cents when commodity costs per pound the amount placed above it.

Example.—When apples, dried, cost 15 cents per pound, for 5 cents one would receive 439 calories, and when they cost 16 cents per pound, for 5 cents one would receive 416 calories. If the price of dried apples should drop to 10 cents per pound, it will be readily seen that one would get twice as many calories for 5 cents as are obtainable when the price is 10 cents per pound. In like manner, if the price were to go to 32 cents per pound, one would get only one-half as many calories as would be received as when dried apples are selling for 16 cents per pound.

TABLE I

A. Apples, dried	15	16	18	20	24	26	28	30	
5 cents = calories	439	416	366	330	275	253	235	219	
Apricots	20	22	25	28	30	35	38	40	
5 cents = calories	315	286	252	225	210	188	173	158	
B. Bacon, smoked	35	40	45	50	55	60	65	70	
5 cents = calories	371	325	289	260	236	216	199	185	
Barley, pearled	08	09	10	11	12	13	14	16	
5 cents = calories	1006	894	805	732	671	619	575	503	
Bass	22	27	32	37	42	44			
5 cents = calories	49	40	34	29	26	25			
Beans, dried Navy.	20	22	25	30	35	38	40		
5 cents = calories	401	365	321	268	229	211	201		
Beans, dried lima	17	20	22	24	30	32	34		
5 cents = calories	478	406	369	339	271	254	239		
Beans, canned baked	25	30	35	40	45	50			
5 cents = calories	120	100	86	75	67	60			
Beans, canned lima.	18	20	22	28	30	33	35	36	Weight of
5 cents = calories	100	90	82	64	60	55	51	50	solid veg.cal.
Beans, canned string	18	20	22	25	28	32	34	36	
5 cents = calories	26	24	22	19	17	15	14	13	
Beef, corned	22	24	28	32	36	40	44		
5 cents = calories	283	259	222	195	173	156	141		
Beef, dried	40	45	50	55	60	70	80		
5 cents = calories	95	84	76	69	63	54	48		
Beef, flank	16	18	20	22	24	26	28	32	Compare
5 cents = calories	345	307	276	251	230	213	197	173	with plate and brisket
Beef-liver	15	18	20	22	24	26	28	30	
5 cents = calories	180	149	135	122	112	103	96	90	
Beef, plate & brisket	16	18	20	22	24	26	28	32	Compare
5 cents = calories	452	403	362	327	306	279	259	226	with flank— both used for goulash
Beef, porterhouse	35	40	45	50	55	60	65	70	
5 cents = calories	157	138	122	110	100	92	85	79	
Beef, round	25	30	35	40	45	50			
5 cents = calories	178	148	127	111	99	89			
Beef, rump	20	25	30	35	40				
5 cents = calories	273	218	182	156	136				
Beef, sirloin	30	35	40	45	50	55	60		
5 cents = calories	163	139	122	108	98	89	82		
Beef tongue	32	36	40	45	50	55	60	64	
5 cents = calories	156	139	125	111	100	91	83	78	
Beets	05	06	07	08	09	10			
5 cents = calories	160	133	114	100	89	80			
Bread, Graham	07	08	09	10	11	12	13	14	
5 cents = calories	309	271	241	217	197	180	167	155	
Bread, white wheat.	07	08	09	10	11	12	13	14	
5 cents = calories	839	734	652	587	533	489	452	419	

TABLE I—*Continued*

Bread, whole wheat.	10	11	12	14	16	18	20		
5 cents = calories	558	507	465	398	348	310	279		
Bread, rye	07	08	09	10	11	12	13	14	
5 cents = calories	824	721	641	577	524	480	433	412	
Butter	45	50	55	60	65	75	85	90	
5 cents = calories	388	349	317	291	268	232	205	194	
C. Cabbage	10	15	20	25	30	35	40		
5 cents = calories	83	55	41	33	28	24	21		
Carp	15	16	18	20	22	24	30		Purchased
5 cents = calories	82	77	68	61	56	51	41		alive
Cheese, Amer. dairy	38	40	42	44	46	48	50	1.00	
5 cents = calories	262	249	237	227	217	208	199	131	
Cheese, Domes, Swiss	60	65	70	75	80	85	90	1.20	
5 cents = calories	162	150	139	130	122	115	108	81	
Cheese, Neuchatal .	30	35	40	45	50	55	60		
5 cents = calories	248	212	186	165	149	135	124		
Chocolate	40	45	50	55	60	65	70	80	Unsweetened
5 cents = calories	347	308	277	252	231	213	198	179	
Cocoa	40	45	50	55	60	65	70	80	
5 cents = calories	282	251	226	205	188	174	161	141	
Cocconut	10	12	15	20	25	30			
5 cents = calories	685	570	456	342	274	228			
Cod-fish steak	22	27	32	37	42	44			
5 cents = calories	76	62	52	45	39	38			
Corn, canned	14	16	18	20	22	24	26	28	Weight of
5 cents = calories	159	139	124	112	101	93	86	79	solid veg.
Corn flakes	20	22	25	28	30	34	36	40	
5 cents = calories	408	371	327	291	272	239	227	204	
Cornmeal	07	08	09	10	11	12	13	14	
5 cents = calories	1152	1008	891	807	733	672	620	576	
Corn starch	10	12	14	16	18	20			
5 cents = calories	816	680	583	510	453	408			
Cottonseed oil	40	45	50	55	60	65	70	80	
5 cents = calories	924	822	740	672	616	569	528	462	
Cream	35	37	40	45	50	55	60	70	
5 cents = calories	252	238	220	196	176	160	147	126	
E. Eggs	42	45	50	55	60	65	70	84	Price per
5 cents = calories	109	101	91	83	76	70	65	55	doz.
F. Fowl, chicken	28	33	38	43	48	53	56		
5 cents = calories	137	116	101	89	79	72	69		
Goose	28	30	35	40	45	50	56		Price, Dec.,
5 cents = calories	263	246	211	184	164	148	132	1916	
Turkey	35	40	45	50	55	60	65	70	Price, Dec.,
5 cents = calories	161	133	118	106	96	88	82	76	1916
Flour, ground wheat	09	10	11	12	13	14	15	18	
5 cents = calories	891	802	729	668	617	573	534	446	
G. Gelatine	1.20	1.25	1.30	1.35	1.40	1.45	1.50	2.40	
5 cents = calories	69	66	64	62	59	57	55	33	

TABLE I—*Continued*

H. Ham, smoked	30	35	40	45	50	55	60		
5 cents = calories	273	234	204	182	164	149	136		
Halibut, smoked ...	28	33	38	43	48	53	56		
5 cents = calories	169	143	124	110	98	89	85		
Hominy	04	05	06	07	08				
5 cents = calories	2010	1608	1340	1149	1005				
I. Ice cream	50	55	60	65	70	75	80	1.00	
5 cents = calories	144	131	120	111	103	96	90	72	
L. Lamb	25	30	35	40	45	50			
5 cents = calories	193	161	138	121	107	97			
Lard	20	22	25	28	30	33	35	40	
5 cents = calories	1021	972	928	887	850	816	785	510	
Lard compound	20	21	22	23	24	25	26	40	
5 cents = calories	405	368	324	289	270	253	238	203	
Lentils, dried	20	22	25	28	30	32	34	40	
5 cents = calories	1021	926	816	729	680	618	583	510	
M. Macaroni	20	22	25	30	32	35	38	40	
5 cents = calories	406	369	325	271	254	232	214	203	
Milk, dry or concen-									
trated	20	25	30	35	40	45	50		
5 cents = calories	100	80	67	57	50	44	40		
Milk, whole	09	10	11	12	13	14	15	18	
5 cents = calories	376	338	307	282	260	241	225	188	
Mutton, leg	25	30	35	40	45	50			
5 cents = calories	178	148	127	111	99	89			
Mutton, loin	28	33	38	43	48	53	58		
5 cents = calories	253	214	186	165	147	133	122		
N. Nuts, almonds	20	22	24	26	28	30	35	40	
5 cents = calories	404	369	336	310	288	269	231	202	
Brazil nuts	25	28	30	32	35	40	45	50	
5 cents = calories	319	285	266	246	228	199	177	160	
Chestnuts	15	18	20	22	25	28	30		
5 cents = calories	307	256	230	209	184	164	154		
Peanuts	10	11	12	13	14	15	20		Unshelled
5 cents = calories	939	850	782	722	670	626	469		and roasted
Pecans	30	35	38	40	45	50	55	60	
5 cents = calories	298	255	227	224	199	179	163	149	
Walnuts	25	28	30	32	35	40	45	50	Domestic
5 cents = calories	172	151	143	134	123	109	95	86	
O. Oatmeal	05	06	07	08	09	10			
5 cents = calories	1810	1508	1293	1131	1006	905			
Olives	45	50	55	60	65	70	75	90	
5 cents = calories	222	200	181	166	154	143	133	111	
Olive oil	55	65	70	80	90	1.00	1.05	1.10	
5 cents = calories	671	569	528	462	411	370	352	335	
Oleomargarine	25	27	30	32	35	40	45	50	
5 cents = calories	682	631	568	533	487	426	379	341	
Oysters, cost per 100	1.00	1.05	1.10	1.15	1.20	1.25	1.30	2.00	Approx. 18
5 cents = calories	91	86	82	78	75	71	68	46	oysters weigh 1 lb.

TABLE I—*Continued*

Onions	08	10	12	14	16	18	20		
5 cents = calories	141	113	94	80	70	63	56		
P. Parsnips	06	07	08	09	10	11	12		
5 cents = calories	192	164	144	128	115	105	96		
Peaches, dried	20	22	25	28	30	35	38	40	
5 cents = calories	315	286	252	225	210	188	173	158	
Peanut butter	20	25	30	35	40				Includes oils
5 cents = calories	706	565	471	404	353				
Peanuts, unshelled .	10	11	12	13	14	15	20		Roasted
5 cents = calories	939	850	782	722	670	626	469		
Peas, canned	15	17	19	21	23	25	27	30	Weights of
5 cents = calories	85	75	67	61	55	51	47	43	solid veg.
Peas, dried	18	20	22	25	30	32	34	36	
5 cents = calories	460	414	370	331	276	259	243	230	
Potatoes, sweet	06	08	10	12	14	16	18		
5 cents = calories	367	275	220	183	157	138	122		
Potatoes, white	07	09	11	13	15	17	20	21	Compare
5 cents = calories	211	164	134	113	98	87	74	70	with rice
Pork, salt fat	30	32	36	40	45	50	55	60	
5 cents = calories	593	555	493	444	395	356	323	297	
Prunes	15	16	18	20	24	26	28	30	
5 cents = calories	387	363	623	290	242	223	207	194	
R. Raisins	20	22	25	30	35	40			
5 cents = calories	401	365	321	268	229	201			
Rice, polished	14	16	18	20	22	24	26	28	Compare
5 cents = calories	568	497	445	401	362	331	306	284	with white potatoes
S. Salmon, canned ...	36	40	45	50	55	60	65	72	
5 cents = calories	140	126	112	101	91	84	77	70	
Sardines, canned ..	16	18	20	24	28	30	32		In oil
5 cents = calories	298	265	239	199	171	160	149		
Sauerkraut	10	11	12	15	16	17	20		
5 cents = calories	63	56	52	42	39	37	32		
Sausage, bologna ..	28	30	35	40	45	50	54	56	Price, May,
5 cents = calories	203	189	162	142	126	113	105	101	1917
Sausage, frankfurt.	25	28	30	33	35	40	45	50	Price, May,
5 cents = calories	227	203	189	172	162	142	126	114	1917
Sugar, granulated .	08	09	10	11	12	13	14	16	
5 cents = calories.	1134	1008	907	825	756	698	644	567	
T. Tapioca	07	08	09	10	11	12	13	14	
5 cents = calories.	1149	1005	893	804	731	670	618	575	
Tomatoes, canned ..	09	10	11	12	13	14	15	18	
5 cents = calories	57	52	47	43	39	37	34	29	
Turnips	04	06	08	10	12	14	16		
5 cents = calories	150	100	75	60	50	43	38		
W. Wheat, toasted	15	17	20	22	24	26	28	30	
5 cents = calories	565	498	424	385	353	325	303	283	
Wheat, shredded ...	10	12	15	20	25	30	35		
5 cents = calories	829	690	552	414	331	276	237		

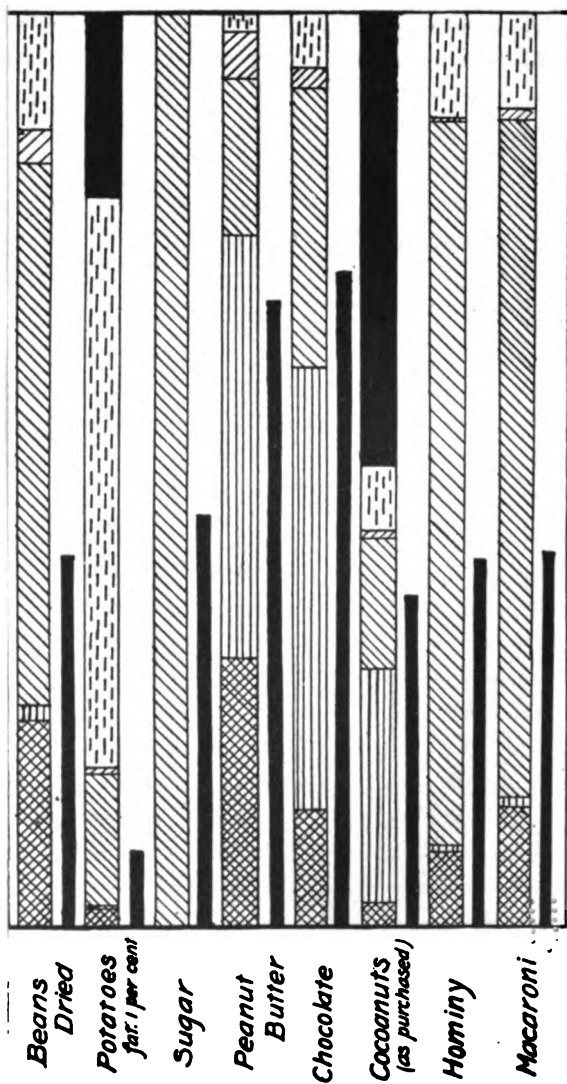
Table I brings out clearly the fact that, while the calories of a dozen eggs or of a pound of beef always remain the same, the number of calories of each of these foodstuffs which one receives for a fixed unit of exchange—say five cents—is entirely dependent upon their market price. In the instance of dried navy beans, which are now selling for twenty cents a pound, one receives for five cents 402 calories. If, however, the price should go to forty cents a pound, one would get only half as many—201 calories.

Table I does not give an idea of the percentage of fat, protein, and carbohydrate in the various foodstuffs. To supply this information, Table II is presented, which shows graphically the fuel value per pound, as well as the percentages of the nutrient constituents—fat, protein, and carbohydrate—of a number of representative foods.

Professor Chittenden, of the Sheffield Scientific School of Yale University, by virtue of numerous experiments, has shown that the amounts of protein necessary to support life under varying conditions, ranging from rest in bed to hard manual labor, are considerably smaller than has heretofore been thought. Indeed, the reports received from the German Army during the earlier days of the war seem to indicate that health may be maintained on protein rations even lower than sixty grammes, or two ounces, dry weight, per diem. The Latin races are the most economical of all peoples in their protein consumption. By the use of large quantities of carbohydrates fortified with oils, which they use in abundance, they derive their energy and heat from these two food elements, and eat merely enough protein material to make up the deficiency caused by tissue waste. In other words, they use protein for repair, and not for energy and heat, deriving their energy and heat almost wholly from fats and carbohydrates.

Table III (p. 271) shows the number of calories in protein, fats, and carbohydrates necessary to support life during varying conditions of activity.

By combining the data furnished in Tables I, II, and III, I believe that information may be secured that will give a better understanding of the value of foods, as they are purchased from the grocers' shelves or as they appear on the table, than it has hitherto been possible to obtain from any one source of information.



Percentage composition and fuel of milk, milk products and some other foods. The constituents are expressed in per cent., the fuel values in calories per pound.

TABLE III
Rations Varied for Age, Sex, and Condition

	Protein		Fats	Carbohydrates.		Energy in calories
	Low	High		Low	High	
Children, two to six	144	230	360	1000	1300	1520-1956
Children, six to fifteen	200	300	405	1300	1400	1923-2123
Women, with light exercise	200	320	720	1200	1320	2272
Women, at moderate work	240	368	720	1600	1728	2720
Man at light indoor work	240	400	540	1560	1800	2764
Man at light outdoor work	240	400	900	1600	1840	2940
Man at moderate outdoor work.	300	500	1125	1800	2000	3475
Man at hard outdoor work	400	600	1350	3000	2200	4000
Man at very hard outdoor winter work	500	720	1800	2400	2600	4902
U. S. Army rations	256	424	2520	1840	2160	4896-5032
U. S. Navy rations		572	2628	2228		5545
Football team (old régime)....		724	2628	2228		5697
College football team (new)....	500	500	1125	2000		3675

I wish here, however, to call attention to certain erroneous conclusions that may be drawn from Table I:

One might, for instance, well consider spinach, celery, lettuce, and various vegetables of this type as a poor investment, because of their low yield of calories. The interpretation of a table of this kind can not be made too literally, since one must consider the important part played in digestion by the gratification of one's fancy or appetite. Lusk, in his introduction to Gephart's book, "Analysis and Cost of Ready-to-serve Foods," makes the following comment: "It is known to all that the sight of appetizing food makes one's 'mouth water.' It is known, however, to comparatively few that the sight of such food makes the stomach 'water' in the same sense; that is to say, a flow of gastric juice is set up in the stomach." Again, he aptly says: "The appetite is like a magic wand, influencing the whole of the digestive process"; and, while many of these food commodities may be a poor investment purely from a caloric standpoint, they must be used in order to make the dietary more attractive. The purpose of Table I is not to show the deficiencies in calories in such foods so much as it is to show, in the main, the types of foods that give one the most for one's money.

Another excuse for using certain of the vegetables that are low in their food value is the mineral elements that they supply. We must not merely consider fats, protein, and carbohydrates when we

think of a balanced ration; for a ration may be well balanced, so far as these constituents are concerned, and very poorly balanced from the standpoint of inorganic constituents. The work of Blatherwick has made it plain that there must be sufficient basic, or alkaline, elements in the diet to neutralize the acidity caused by chlorine, phosphorus, sulphur, etc., which are acid-forming elements, and which preponderate in meats and cereals. These substances would be instrumental in raising the hydrogen-ion concentration of the serum, were it not for the basic elements referred to, which predominate in most vegetables.

Table IV shows the preponderance of either acid or basic elements in a group of foods. For instance, while lemons and straw-

TABLE IV
(Sherman and Gettler) *

Excess of Acid-forming or Base-forming Elements in Foods †

Article of food	Excess acid or base in terms of normal solutions. Per 100 grammes.	
	Acid Co.	Base Co.
Apples		3.76
Asparagus		0.81
Bananas		5.56
Beans (dried)		23.87
Beans (lima, dried)		41.65
Beets		10.86
Cabbage		4.34
Cantaloupe		7.47
Carrots		10.82
Cauliflower		5.33
Celery		7.78
Crackers	7.81	
Eggs	11.10	
Egg-white	5.24	
Egg-yolk	26.69	
Fish (haddock)	16.07	
Lemons		5.45
Lettuce		7.37
Meat (lean beef)	13.91	
Milk (cow's)		2.37
Oatmeal	12.93	
Oranges		5.61
Potatoes		7.19
Prunes		24.40
Raisins		23.68
Rice	8.10	
Wheat (entire)	9.66	

*SHERMAN AND GETTLER: *Jour. Biol. Chem.*, 11, 323, 1912.

†HAWK: "Practical Physiological Chemistry," Fifth Edition.

berries are acid, the principal part of their acidity is due to such acids as citric acid, which contain neither acid nor basic elements, and are broken up into carbon dioxide and water. Lemons have, as will be seen from the table, a preponderance of basic elements, like potassium, sodium, etc., over the acid elements, such as chlorine, phosphorus, sulphur, etc. It is a common practice among physicians to administer alkaline citrates when it is desired to increase the alkalinity of the urine, since these supply available alkaline ions.

Another excuse for employing vegetables that are low in food value is the fact that they are easily fortified with fats and oils in a manner that increases their palatability and greatly enhances their nutritional value.

Still another reason for their use is the fact that they leave a large intestinal residue or roughage, which facilitates a regular function of the bowels.

In order that one may more readily gain an approximate idea of the caloric value of his intake, Table V is added, giving, in one hundred-calories portions, the number of teaspoonfuls, tablespoonfuls, etc., of prepared or ready-to-eat foods.

There are 100 calories in the following:

TABLE V

Oyster crackers	20	1 inch in diameter
Uneda biscuits	4	
Homemade bread	1	slice, 3 x 4 x $\frac{1}{2}$ inches
Flour	5	heaping tablespoonfuls
Oatmeal, cooked	2 $\frac{1}{2}$	heaping tablespoonfuls
Hominy, boiled	2 $\frac{1}{2}$	heaping tablespoonfuls
Macaroni, boiled	2 $\frac{1}{4}$	heaping tablespoonfuls
Macaroni, baked with cheese..	1 $\frac{1}{2}$	tablespoonfuls
Flour, roller process	1	tablespoonful
Corn starch	1	tablespoonful
Rice, boiled	1	heaping tablespoonful
Potatoes	1	very large
Sugar, cubes	3 $\frac{1}{2}$	
dominoes	4	
loose	2 $\frac{1}{2}$	heaping teaspoonfuls
Cornmeal mush	4 $\frac{1}{2}$	tablespoonfuls
Butter and oleo	1	ball
Olive oil	1	scant tablespoonful
Cheese, American pale	1	cubic inch
Eggs, hen's	1	large
Milk	1 $\frac{1}{4}$	tumblerfuls
Bacon, smoked, uncooked	$\frac{1}{2}$	slice

TABLE V—*Continued*

Mutton, lean	1	small chop
Steak, round	1	small helping, less than 2 ounces
Oranges	1	large
Tomatoes	3	
Bananas	1	
Peaches	3	
Prunes	2	very large
Figs	1½	
Walnuts	3	large

When Joslin mentioned the part played by tuberculosis in revolutionizing therapeutics, he failed, in my opinion, to point out one of the most important and far-reaching things that tuberculosis has given us; namely, the therapeutic value of rest. In the treatment of tuberculosis, rest is one of the most essential factors. Permit me, therefore, in concluding this article, to leave with you a thought that I have expressed elsewhere in the daily press:

It requires less fuel to run an internal-combustion engine fourteen hours than it does to run it sixteen hours. In like manner, it requires less fuel to run the human engine fourteen hours than it does to run it sixteen hours. Now, it is a conservative estimate that the average human being will save one hundred calories by going to bed two hours earlier at night. If thirty-five millions of the more than one hundred millions of people in the United States were to make such a daily conservation for one year, it would be ample to feed an army of one million men for two hundred and fifty-five days.

It is not to be thought that my expectation is that this statement shall be taken literally. I have presented my idea in this peculiar manner in order to bring forcibly home to the minds of my readers the therapeutic value of rest, at a time when food may be scarce, and in order that tuberculosis and other pestilences that have already seized the countries that are now so vigorously engaged in warfare be prevented. With increased rest and increased relaxation, one may not only accomplish these purposes, but may also be enabled to work more intensively at one's occupation during the hours given to such work.

In this connection it is to be hoped that there will be a nationwide movement toward the establishment of a curfew, which would mean not only a conservation of food, clothing, leather, etc., but it would be highly instrumental in production of a sturdier and more disease-resistant race.

Surgery

TREATMENT OF PYÆMIA AND SEPTICÆMIA BY TRANSFUSION OF IMMUNIZED BLOOD

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SEPTICÆMIA is a febrile condition due to the absorption from a wound into the blood-stream of pyogenic bacteria and their toxins. Owing to lack of natural resistance on the part of the infected individual, the bacteria are not destroyed by the plasma and leucocytes, but multiply in the blood-stream. The condition is spoken of as pyæmia when the circulating bacteria increase to such an extent that masses of them plug the capillaries and form septic thrombi which break down into local abscesses. Pyæmia is an advanced septicæmia characterized by the formation of secondary or metastatic abscesses.

VARIETIES OF SEPTICÆMIA

Clinically septicæmia falls into three general types: *First*, an acute localized cellulitis and lymphangitis, giving general febrile symptoms, which subside with free drainage of the infected area. The bacteria in the blood-stream are quickly overcome, once their source of supply is cleaned out.

Second, the acutely fatal type, in which the primary focus is of secondary importance. The bacteria which have entered the blood-stream multiply rapidly and in spite of all treatment the individual is overcome in from five to ten days. The toxæmia is so virulent and its action so rapid that local abscesses do not have time to form before death occurs.

Third, the true pyæmia, in which there is an attempt at localization, on nature's part, of the circulating bacteria. They are walled off at various spots in the body and form abscesses, particularly in the skin, bones, and solid viscera. The patient runs his course of fever, sweats, chills, vomiting, septic diarrhoea, enlarged spleen, jaundice,

albuminuria, progressive anæmia, and delirium, and finally succumbs in from three to six weeks' time.

In the first type of septicæmia local measures suffice; in the second type the case is fatal from its incipency; in the third type materials thrown into the blood-stream which will kill the circulating bacteria without injuring the host may effect a cure. Pyæmia is a progressively fatal disease when treated by the usually accepted methods. It is against this type of infection that the efforts of workers in the fields of chemotherapy and serumtherapy are now being directed.

GENERAL TREATMENT

Locally.—Care of the infected focus by thorough drainage and antiseptics. This, while important in the early stages, is of little value after the infection is general and the blood-stream has become the body culture media. Later, every metastatic abscess should be opened and drained in the hope of preventing its becoming a secondary source of infection.

Medication by Mouth.—Almost every drug in the Pharmacopœia has been given in the hopeless search for a specific—notably iron, quinine, alcohol, and the mineral acids. All are useless except for their general tonic effect.

Intravenous Medication.—Theoretically this offers the best method of attacking the infection. All antiseptics which have been successfully used in local conditions have been tried in safe dilution intravenously. Mercury, carbolic acid, formalin, and turpentine (thallanine) have had enthusiastic exponents, and, while an occasional case may have reacted brilliantly, the real chemical specific has not been found. Proteins as derived from starch solution, Witte's peptone, or the bacterins given intravenously are said to break down the infective body foci and increase the bactericidal action of the blood. Substances tending to neutralize the blood acidity in acidosis states, as glucose and soda bicarbonate (Fischer's solution), have value when injected in salt solution and may prolong life, but the end-result is the same.

Vaccines or bacterins are useless. The patient's body is in a continuous negative phase, and the throwing into it of some millions of dead germs but adds more fuel to the fire of the countless living organisms which the system is unable to react to.

Sera—the most exploited of which is the antistreptococcic—is of no proved value.

Elimination, by purging, sweating, and diuresis, supplemented by salt solution intravenously or subcutaneously, by diluting and carrying off the toxins gives immediate good results, which unfortunately are only temporary, and the day arrives when the toxins of the multiplying bacteria can no longer be washed away.

Hypodermic Stimulation.—Strychnia, digitalis, caffein, etc., like salt solution, have no permanent value.

This whole list of remedies, while giving temporary results, is useless in checking a fatal termination of the disease, because they treat the toxins and not the germ which is producing them, and until the workers in chemotherapy can give us drug combinations to kill each strain of blood-circulating organism, similar to the action of salvarsan on the *Spirochæta pallida*, we will have no exact method of combating septicæmia and pyæmia.

TRANSFUSION OF IMMUNIZED BLOOD

The use of whole blood in the treatment of septic conditions has been practised by many operators with indifferent success. It is probable that the blood elements of the usually employed, normal, healthy donor are quickly destroyed by the toxins and bacteria of the recipient, and at the best can act only as a fleeting reinforcement. Indeed, such rapid destruction as indicated by the chill of hæmolysis may accentuate the existing toxæmia.

But if the transfused blood is loaded with protective principles; if the donor has recently cured himself of a similar infection, his serum must be strongly bactericidal and his leucocytes trained to combat that type of infection from which he has recovered and from which the recipient is dying.

With this idea in view, every progressive case of septicæmia or pyæmia in the Samaritan Hospital is given this treatment exclusively, with results which have been most encouraging. The eight cases reported tell their own story. All were of the progressively fatal type under usual methods of treatment. Two had been refused admission to other hospitals because of their hopeless condition. Seven of the eight made complete recoveries. The fatal case responded to his first transfusion, but died of a pneumonia which developed three days later.

RECORD OF CASES TRANSFUSED

	History	Clinical symptoms	Donor	Transfusions	Result
I. R. C. PYÆMIA	Female, age 31 years. Palmar abscess treated by incision, irrigation and Bier hyperæmia. Chill on 17th day.	Fever, sweats, chills, abscesses of buttocks, axilla and groin, large spleen, jaundice, diarrhoea. W. B. C. 11,300 Hæmg 38 per cent.	Recovered from streptococcal infection of leg three weeks before.	I. 8 os., on fifth day of pyæmia. II. 8 os., on seventh day. III. 8 os., on eleventh day. IV. 8 os., on sixteenth day.	Recovered.
II. H. S. PYÆMIA	Female, age 18 years. Tonsillitis followed in two weeks by septic arthritis. Pus showed colon and staphylococci.	Fever, sweats, chills, abscess of right ankle. Fluctuating left ankle and wrist. Abscesses of buttocks; enlarged spleen. W. B. C. 18,000 Hæmg 60 per cent.	Recovered from a colon and mixed staphylococcal cystitis and prostatitis two weeks ago.	I. 8 os., in second week of pyæmia. II. 4 os., one week later.	Recovered.
III. E. A. SEPTICÆMIA	Male, age 10 years. Uncontrollable supuration following crush of leg two months ago. Sequestra removed.	Hectic temperature. Albuminuria, diarrhoea, large spleen. Delirium. W. B. C. 16,000 Hæmg 25 per cent.	Recovered one month from an infected compound fracture of the leg.	I. 6 os. II. 4 os., one week later.	Recovered, useful leg.
IV. W. H. PYÆMIA	Male, age 25 years. Infected compound fracture six weeks ago.	Fever, sweats, chills, phlebitis, abscesses of buttocks, arthritis of wrist. W. B. C. 15,000 Hæmg 60 per cent.	Recovered six months from an infected compound fracture of the leg.	I. 10 os. II. 8 os., five days later.	Recovered.
V. M. S. PYÆMIA	Male, age 50 years. Car-buncle of back of three weeks standing. No glycosuria.	Fever, sweats and chills, phlebitis; abscess of thigh; delirium. W. B. C. 9,500 Hæmg 60 per cent.	Recovered three months from a severe general furunculosis.	I. 8 os. II. 8 os., five days later. III. 8 os., eight days later.	Recovered.
VI. T. W. SEPTICÆMIA	Male, age 28 years. Abscess of lachrymal sac followed by spreading cellulitis of face and neck, of one week's standing.	Chills and fever, delirium. W. B. C. 22,000	Recovered three weeks from a streptococcal infection of leg.	I. 12 os.	Local and general conditions improved. Pneumonia developed third day after transfusion from which patient died.
VII. H. W. SEPTICÆMIA	Male, age 26 years. Palmar abscess of five weeks' standing. Spreading cellulitis and lymphangitis of arm and shoulder.	Chills and fever, delirium, enlarged spleen, septic diarrhoea. W. B. C. 14,600 Hæmg 68 per cent.	Recovered three weeks from a cellulitis of scalp and erysipelas of face, neck and chest.	I. 8 os. II. 6 os., one week later. III. 6 os., five days later.	Recovered.
VIII. C. R. PYÆMIA	Male, age 23 years. Infected wound of knee nine weeks ago resulted in a deep and superficial cellulitis of leg.	Fever, sweats and chills, septic arthritis knee and hip. Supporting sinuses of leg, multiple skin abscesses. Enlarged spleen. Albuminuria. W. B. C. 9,000 Hæmg 38 per cent.	Recovered three weeks from infection of hand and arm.	I. 8 os., two days before a hip-joint amputation. II. 8 os., three days after amputation. III. 8 os., one week later.	Recovered.

Plan of Treatment.—A record is kept of all recovered septic cases, particularly streptococcic. Cultures or smears are immediately taken of any resistant septicæmic or pyæmic case which develops in the house or is admitted in this condition. A donor is chosen whose type of infection clinically and bacteriologically is similar. From four to twelve ounces of blood is at once transfused. If time and the patient's condition permit, hæmolysis tests are made. The dose is repeated on an average of every fifth day until the patient is symptomatically cured. In two cases with urinary suppression Fischer's solution was given intravenously every second day. In the other six cases no other treatment was given. The immediate improvement in appetite and digestion allows of forced feeding.

The most striking effect of the treatment was the sense of returning confidence and well-being on the part of the patient. Within twelve hours there is a drop in temperature, cessation of sweats and chills, return of normal mentality, return of appetite, a fading away of forming abscesses, and an improvement in local wound conditions, as evidenced by a limitation of spreading cellulitis and a free discharge of normal pus in open wounds. The magic of the disappearance of symptoms is only comparable to similar changes in old mercury-proof syphilitics following an intravenous injection of salvarsan.

Experience has taught that the transfused blood is used up in about five days, and in bad cases symptoms of a relapse may be expected after that time if a second transfusion is not given; so that two doses of blood at five days' interval is the rule, with subsequent doses if the nature of the case demands it. In no instance did symptoms of hæmolysis develop, nor was there evidence of the blood doing the recipient harm.

As the dose of blood is used up within a few days, there is danger of giving too few transfusions instead of too many, and of attempting to throw the patient back on his own body resistance, which does not exist, instead of relying on the positive value of a suitable donor's blood. This fact was well shown in Cases I and V, in which febrile symptoms developed following the patient's discharge from the hospital as cured. The relapses occurred three and four weeks, respectively, after all local and general symptoms had subsided, and relief was only attained by a resumption of treatment. This is a class of

patients who have no autogenous resistance and must depend on outside elements to combat their infection.

The limitations to the treatment are the finding of a suitable and willing donor and the ability to carry out a transfusion. We have found the syringe-cannula method the easiest and most reliable transfusion technic.

The question of how long the immunizing principles remain in the blood of a self-cured septic case is a matter for future research to decide. The blood of one of our donors was still active clinically after six months; but one would expect the best results from the serum and corpuscles of the recent, healthy convalescent.

CONCLUSIONS

1. Transfusion of blood from self-cured septic cases has a curative action on patients suffering from chronic septicæmia or pyæmia.
2. The infection from which the donor has recovered should correspond clinically and bacteriologically to the infection by which the recipient is being overcome.
3. The transfused blood is used up in about five days, and a second dose should be given at that time and repeated as often as the clinical symptoms warrant.
4. The immediate improvement in symptoms is so marked that one gets a false sense of security, which will be shattered by a relapse if repeated transfusions are not persisted in.
5. It offers a promising method of cure in the usually fatal chronic septicæmia and pyæmia conditions.

ETHER ANÆSTHESIA

BY KATHLEEN CARROLL, R.N.

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THE success of any anæsthetic depends upon several factors, the principal among these being as follows:

First.—Its safety.

Second.—Its anæsthetic properties.

Third.—Its after-effects upon the patient.

Fourth.—Its convenience to the surgeon.

Fifth.—Its ease of administration.

From wide experience ether has proved itself to be the safest of all anæsthetics—so much so that in many of the large clinics it is used to the exclusion of all others. Ether is absolutely safe, inasmuch as it may be administered by the most inexperienced person, which unfortunately is too often the case. If the time were given to the study of its proper administration that must necessarily be given to the study of nitrous oxide, many more would appreciate its truly valuable properties as an ideal anæsthetic. Many unfavorable comparisons have been made between ether and nitrous oxide, but upon investigation it was found that these comparisons usually were made by an expert with the latter anæsthetic and an inexperienced person with the former. Nitrous oxide is very often used as an initial anæsthetic, but this is unnecessary if ether is administered properly, as the patient should experience no feeling of suffocation if given plenty of air and the ether is dropped slowly. The use of chloroform as an initial anæsthetic is absolutely unwarranted; furthermore, statistics show dangers attached to its use that prohibit it in many instances. Scientific investigation has demonstrated that it is a direct protoplasmic poison, and no precautions can be intelligently taken against it.

The anæsthetic properties of ether are best demonstrated in the perfect relaxation which may be obtained by its use in abdominal operations. From personal observation it may be stated that where nitrous oxide is used for abdominal operations it is impossible to

obtain perfect relaxation without the use of ether in conjunction with it or where the surgeon is able to work in the abdominal cavity without packing off the intestines. In giving ether where there is work upon the stomach or intestines the anæsthetic may be stopped when the abdomen is opened and the patient allowed to come out almost entirely—ether again being administered before the surgeon commences to close the peritoneum in order that there may be relaxation for that purpose.

The great virtue of nitrous oxide seems to be in its conjunction with novocaine—the anæsthetic being given merely to allay the shock which the patient is apt to experience from witnessing her own operation; but it does not insure relaxation. Thus comparing the use of nitrous oxide and novocaine and ether and novocaine—in many cases where the latter is used, *goitre cases* especially—a very small amount is required to keep the patient under.

The after-effects of ether upon the patient, as heretofore chronicled, have so greatly diminished since its proper administration that it leaves little or nothing to be said upon the subject. Shock following the administration of ether is very uncommon, as likewise is pneumonia, the latter, where it has occurred, having been found to develop in most cases in from a week to ten days following operation, thus eliminating ether as the offending factor. Its effect upon the kidneys is, at the worst, only transitory. This is evidenced, perhaps, by the presence of acetone in the urine, which, however, has been found to clear up very readily in a day or two after the operation.

Ether has been found to be a cardiac stimulant in small doses, and may be administered accordingly in heart cases.

I have stated above the convenience of ether to the surgeon, inasmuch as it is the only anæsthetic that will produce perfect relaxation and enable him to carry on his manipulations without troublesome straining of the patient.

The administration of an anæsthetic is not only a science but an art, and one of the greatest factors in giving an anæsthetic is *to talk the patient to sleep*. Where it is at all possible, a preliminary visit should be made to the patient so as to assure her of the fact that there is no danger in taking an anæsthetic and to gain her confidence. Many people who undergo operations have a far greater dread of the anæsthetic than they have of the operation itself.

The anæsthetic room should at all times be absolutely quiet in order that there may be nothing to distract the patient's attention from the anæsthetist, and, as the mental attitude and feelings of the latter are reflected upon the patient, she should give her undivided attention to the anæsthetic. She should assure the patient that nothing will be done until she is asleep; this seems to be a great dread with so many, and it is much better to wait and encourage a patient—taking great care never to cross them—than to try and force them under. Undue haste usually results in struggling, coughing, and vomiting, and nothing is gained; whereas if a patient is allowed to take her own time and is given plenty of air, narcosis without the above inconveniences is quickly and comfortably acquired.

Ether is best given by the slow drop method, using for convenience a four-ounce can or bottle, into which is fitted a cork, grooved on either side, through one of which a small wick of cotton is passed into the can. A modified Esmarch inhaler, covered with two layers of stockinet, is found to be easy to handle—the inhaler being sterilized by boiling between operations and a fresh piece of stockinet used for each operation. The patient's eyes are protected by a piece of wet cotton being placed over them, under which a piece of gutta-percha tissue may be used, although this is not absolutely necessary. Great care should be taken to remove false teeth, chewing gum, etc., and the nose should be inspected to see that all air-passages are open. The cone is at first held away from the face while the ether is dropped slowly. Many patients will respond more easily if asked to count slowly.

The anæsthetist should acquaint her patients with the different stages of anæsthesia and tell them what to expect, so that as the ether begins to take effect the cone can be lowered to the face and the gauze wrapped around it—always being careful to leave an air-space in order to keep the color good. The jaw should be kept well forward and up so as to keep the tongue from falling back and to insure against vomiting. When the muscles are relaxed, the head should be placed on one side, as patients are found to breathe better in that position. Many anæsthetists make the mistake of doing too much rather than too little, and a more comfortable anæsthesia is obtained and maintained if the patient is disturbed as little as possible—provided they are in good condition. Surgical anæsthesia is

easily recognized by the deep, regular respiration and relaxation of the muscles.

In anæsthesia the color sign is perhaps the greatest register of a patient's condition, and the anæsthetist should be able to recognize the difference between pinkness and pallor as signifying the difference between a well-sustained circulation and a condition of syncope. The greatest endeavor should be made to keep the patient's color pink, as there is always danger when there is pallor or the patient becomes purple; both serve to indicate an insufficient amount of oxygen and are a warning for less rebreathing and more fresh air. Pallor is more apt to occur where a too open method is employed and an excessive loss of carbon dioxide takes place, thus lowering the visceral tonus and the normal amount of blood in the cutaneous vessels: rebreathing in such cases is indicated. Also, with the open drop method, there is much waste of ether, due to evaporation and the annoyance of a dripping frosted mask, and few patients will go under without the anæsthetist having to resort to the closed or semi-closed method, as with the open method vomiting is very apt to occur and the anæsthetic must necessarily be "pushed" to counteract it.

Ether should be given as any other drug, with the same precautions for over-dosage. It is difficult to state any particular rules as to the amount required for any particular operation, as so much depends upon the patient: cases of peritonitis usually, and alcoholics especially, require much more than ordinary abdominal cases; whereas head and neck cases may be operated upon under light anæsthesia, while rectal cases require full anæsthesia. Where novocaine is used, especially in goitre cases, very little ether is required.

Postoperative vomiting following ether is present in a very small percentage of cases: when toxic doses are given and the blood becomes saturated with it, naturally the stomach is called upon to eliminate it, and under these circumstances it is a virtue rather than a fault. A single emesis, which often takes place before return of consciousness, has the advantage of clearing the lungs as well as the stomach.

THE IMMOBILIZATION OF FRACTURED LIMBS IN MILITARY SURGERY

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WITH modern arms whose projectiles are possessed with a very great momentum and explosives which disperse bursting steel in many-sided, sharp-edged bits, fractures in warfare are mostly compound, with large, irregular wounds, numerous bone splinters, and severe suppuration.

Soiled by contact with every possible kind of matter, containing in their recesses débris of clothing, earth, metallic dust, and various other foreign bodies, all these wounds must be considered infected from the start. In September, 1915, Tuffier stated at the Academy of Medicine that 50 per cent. mortality occurred in wounds treated in the ambulance service from infected wounds, and out of 1000 amputations, 800 were rendered necessary by infection.

Therefore these wounds should be the object of minute and unceasing care, for otherwise infection supervenes, suffering is extreme, hemorrhage will reduce the patient's vitality, and, finally, gangrene will necessitate amputation.

But another object to be attained must be ever borne in mind, and that is to obtain a functionally useful limb, and this brings up the question of the conditions which an apparatus for the treatment of the fractures must fulfil.

The above two therapeutic indications can not be separated. Under no circumstances can such fractures be put up in a plaster cast, for obvious reasons, and if, on the other hand, all care is exclusively directed to the treatment of the wound and healing is awaited before applying an apparatus for the treatment of the fracture, it is more than probable that, after the lapse of the several weeks required for the repair of the wound, reduction of the fragments will be a matter of impossibility.

Warfare surgery was confronted with a great difficulty, and from the beginning the treatment of these compound fractures appeared

overpowered by obstacles. Frequent dressings on the one hand, immobilization of the fracture on the other, as much for obtaining consolidation in good position as to spare the patient the suffering resulting from the removal of the apparatus, were urgent necessities.

An apparatus which would permit an easy and frequent examination of the wound, the application of dressings and drains, ease the pain due to displacement of the fractured bone by retaining the fragments in proper position, is, as Cabot says, the *vital indication*.

But, at the same time, the apparatus must be capable of immobilizing the limb after the lapse of several days, giving the patient a limb of nearly normal shape and functionally useful. That apparatus which can give the best ultimate results will fulfil the orthopædic indication.

It should also fulfil other requirements. While controlling the pain inherent to movements, it must not in itself be painful, and it should occupy as little room as possible and be as light in weight as possible over the traumatic area, without ceasing to be sufficiently resistant to maintain the reduced fracture, and prevent overlapping of the fragments. It must be simple, quickly constructed with materials which can not absorb pus, and unattackable by the contact of antiseptic solutions. Such materials are easily obtained.

Finally, the apparatus must be such that it can be applied anywhere and to any case that may come up for treatment.

My studies with various apparatuses made of plaster or other material, at the front in ambulances or in the hospitals in Paris, made evident to me the advantages and disadvantages of each system and at length led to what I consider the ideal type and which gives the best results in this class of fractures. This apparatus is what I call the *bracketed plaster collars*, but before describing it let me say that neither fracture cradles, splints of all types, continued extension, or the usual plaster casts can realize at the same time both therapeutic indications of open fractures; namely, *painless and frequent dressings and immobilization in good position*.

Now, although continued extension permits examination of the wounds, it is not an apparatus with which transport of the patient can be undertaken with any degree of security. With a medium weight it does not avoid, any more than the fracture cradle, the dangers of hemorrhage from injury of the vessels by the ends of the

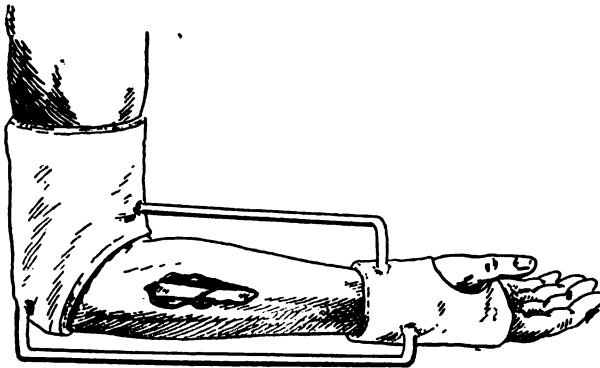
fragments. If a heavy weight is used, which prevents shortening and deformity of the callus, it must be applied over a large segment of the limb and consequently can not be applicable in cases of extensive wounds. Extension is not practical for frequent removal.

The ordinary plaster cast is the best for maintaining approximation of the fracture, but it prevents the local treatment of the wound. Suture of the fracture or bone plating is, in war surgery, a very exceptional means of treatment.

The bracketed plaster cast (that is to say, a combination of rather broad plaster collars and metallic splints) *allows the wounds to be carefully watched at all times and secures immobilization which controls the pain.*

Quénu was the first to use these splints, and the one that he

FIG. 1



Open fracture of forearm. Plaster collars and tin splints applied.

employs for the lower limb realizes continued extension at the same time. It is composed of a plaster cast around the waist and one or two collars on the legs, according to the requirements of the case, united by copper rods which slide in metal tubes fixed in the plaster and to which they are fastened in extension (see Fig. 1).

All these interrupted plaster apparatuses are composed of more or less broad plaster collars applied on the healthy portions of the limb and taking their point of application on the more prominent parts of the skeleton. They are connected by rods of various materials—wood, plaster, or metal. The rods are straight, coaptating themselves to contours of the limb, or are made in the form of a handle or arch in order to give easy access to the wound.

Lejars uses rather heavy iron wire for the rods, while Rosenblith employs lead tubes over which rubber tubing is drawn. I do not recommend wooden rods, for obvious reasons which require no discussion.

Plaster splints take up much room and are quickly soiled, while plaster arches are not sufficiently strong to permit transportation of the patient.

In order to give free access to the wound the rods must be as small and as light as possible, without involving the solidity and rigidity of the apparatus. They must likewise adopt the shape and, so to say, mould themselves on the limb in order to be correctly applied and give the necessary support.

There is only one malleable splinting material which fulfils these requirements, and that is one of metal which does not oxidize, which is not altered by contact with pus and antiseptic solutions, and which at the same time retains sufficient rigidity to maintain the extension when applied.

These rods, as I have said, may be straight, arched, or in the form of bridges, but a fact that must not be lost sight of in the conception of a military fracture apparatus is that, of all its qualities, simplicity and the ease with which it can be constructed are of paramount importance.

A certain number of these apparatuses constructed in most ingenious ways are used in the large base hospitals, but they possess precisely the faults of their qualities—they are not sufficiently practical. At the ambulance or field hospital they are not to be had and are too intricate to construct.

Delbet has recorded some very interesting results obtained with different types of these apparatuses by which continued extension is obtained and walking at the same time permitted. The first of them is composed of tubes which slide into one another, but a spring, whose strength is regulated by screws, forces them apart, thus producing an opposing effective force on the plaster collars to which they are fixed.

In the second type, the weight of the body is supported by the splints by the intermediary of the plaster collars. Still other types,

too long to describe, have given good results at the hands of other surgeons.

Simplicity and ease of construction are the great qualities of the apparatus made with strips of thin sheets of tin, a material which can be found everywhere. It has the great advantage of moulding itself to the shape of the limb that it is to support. The application of these narrow strips of tin to the plaster requires very little time; they are thin, light, take up very little space, are *rigid*, and *do not oxidize*.

By this combination of plaster and tin splints a sufficiently rigid apparatus is obtained to retain the limb in proper position, and consequently the fragments can not become displaced. The pain ceases and the patient can sleep with comfort. This is one of the most appreciable results obtained as soon as this apparatus has been applied, while the easy access to the wound, permitting frequent change of dressings and the resulting relaxation of the muscles, makes it an ideal splint.

Quite as much as the more complicated types of fracture splints, it facilitates continued extension and transportation of the patient.

The ultimate results are excellent, and the cases under my observation, both in ambulances and hospital wards, prove that these apparatuses are quite sufficient to retain the fragments and limb in good position. As a return of the functions of the limb is a capital point in treatment, it is important to note that in this respect our results have been excellent.

I will here give some notes of cases taken at random.

CASE I.—Y. C., aged twenty-one years, wounded by bursting shell on November 13. Fracture of the head of the right fibula. November 21, extraction of the projectile. December 7, incision over patella and on the inner side. December 23, posterior incision. The wound suppurated freely and the patient failed rapidly. From January 6 to 8 violent pain was constantly present and the general health was very poor. January 9, curetting of the granulation tissue, incision of a vast purulent collection at the posterior aspect of the joint, exposure and removal of enormous bone splinters of the head of the fibula. Declivous drainage of the popliteal space. January 15, application of two plaster collars above and below the knee-joint, united by two strips of tin. The next day the pain was less, and

two changes of dressings were made daily. February 1, after a period of great weakness with diarrhoea, thrush, and bilateral parotiditis, the patient recovered with a knee in excellent condition.

CASE II.—H. M., aged thirty-eight years, wounded January 4 by a bursting shell. Entered hospital January 10, discharged March 3. Comminuted fracture of right femur by a ball which entered in front and below the great trochanter. Incision and an external counter-incision, removal of bone splinters, drainage.

February 3, wounds in excellent condition, application of a plaster waistband extending downward in the form of a band over the anterior aspect of the thigh and joining a plaster collar at the posterior aspect by turning over the internal aspect of the thigh. A tin splint on the external aspect joined the waistband with a plaster collar applied below the knee. The frequent changes of dressing were easily made. Patient discharged with the limb in excellent condition on March 3.

CASE III.—R. G., aged twenty-eight years, wounded December 20 by bursting shell. Entered hospital on December 25, discharged February 28.

Compound fracture of right forearm, multiple bone splinters and strips of periosteum which were left in place; cleaning of the wound and drainage. Comminuted fracture of left humerus at the two lower thirds. Fracture of both bones of the left forearm at their upper third. Curettage, application of a tin splint placed on the inner aspect of the elbow-joint and retained by two plaster collars, one at the wrist, the other above the elbow, and a third above the biceps. Dressings painless and easily changed.

Patient discharged in good condition on February 28.

CASE IV.—J. M., wounded December 15, entered hospital December 25. Joint fracture of left knee. January 18, suppuration continuous, the joint was enormous and fluctuating, temperature 39.5° C., severe pain. External arthrotomy giving issue to a large quantity of purulent fluid. Application of two plaster collars, one above, the other below the knee, and joined by two arched tin strips.

February 7, incision of two foci under and above the joint. February 8, dressings changed, wound in good condition. February 11, anterior, posterior, and internal incisions made; the suppuration

continued in spite of the free opening and drainage, so that amputation was done on March 2.

CASE V.—T. R., aged thirty-one years, wounded December 24 by rifle bullet. Entered hospital December 28, discharged February 28.

Fracture of the head of right humerus. December 27, exploration under chloroform narcosis showed an upper fragment, appearing to be the head of the humerus, and a fragment about two centimetres distant from it, with detached splinters of bone. Slight supuration.

From January 17 to 20 the temperature rose progressively. January 21, exploration under chloroform: head of humerus black and partially necrosed. Application of plaster splint composed of a collar surrounding the neck and a portion of the upper part of the shoulder, and another holding the forearm in a right angle by means of two tin strips joining the two plaster collars. January 22, dressings changed, wound in very good condition. No pain.

Patient discharged on February 28.

CASE VI.—A. D., aged twenty years, wounded January 20 by a shrapnel bullet. Entered hospital January 21.

Fracture of right femoral condyles. Bullet entered inwardly and in front of the external condyle; wound of exit large at the level of the internal condyle. Incision and removal of numerous bone splinters. Complete destruction of internal condyle; the external condyle was chipped. Application of a plaster splint composed of two collars, one on the thigh, the other on the leg, extending down to the instep. These collars were joined by two strips of tin on the internal and external aspects of the limb. Operation progress good.

February 2, pain in the calf, rise in temperature. The apparatus was removed and purulent tracts were discovered, one in Hunter's canal, the other extending along the femur. The external condyle had become completely detached with a portion of the femoral diaphysis. Odor very strong. Resection of the knee-joint was attempted, but the shortening was so great that amputation was done.

CASE VII.—P. C., aged nineteen years, wounded January 8 by gunshot. Entered hospital January 10, discharged February 19.

Fracture of the thigh in the subtrochanteric region. The upper fragment overlapped as soon as traction of the leg was stopped.

Limb immobilized in Tillaux's apparatus after disinfection of the focus of fracture.

February 8, a plaster waist was applied with an under-thigh extension, a plaster collar below the condyles and another above the malleoli, with tin strips uniting them. All pain ceased.

Discharged on February 17 in good condition.

CASE VIII.—A. L., aged twenty-one years, wounded January 19 by gunshot. Discharged February 26.

Fracture of the thigh. Penetrating wound. Entrance opening on the external posterior aspect above the knee, orifice of exit within and above the knee. Incision of the latter, at the bottom of which a mass of small bone splinters was seen and removed. Application of a plaster collar surrounding the upper and middle thirds of the thigh and another on the leg, united by tin strips.

Dressings were painless. Patient discharged in good health on February 26.

CASE IX.—H. P., aged twenty-two years, wounded January 22 by a bullet. Entered hospital January 24. Had been operated on at St. Menchould, where a complete section of the half of the arm situated behind the wound had been done.

The radial nerve was exposed. The bone splinters were removed and both ends of the fractured humerus were evened off with the saw.

January 25, a plaster collar was applied round the neck and another to the forearm; these were joined by tin strips, maintaining the arm at a right-angle.

January 30, the infection had progressed considerably, so that amputation was done.

CASE X.—P. R., aged twenty-six years, wounded January 22 by rifle bullet. Entered hospital January 24.

High compound fracture of right thigh. Wound freely opened. Plaster waistband and three collars united by tin strips.

March 7, radiography showed that the head of the femur had completely disappeared.

April 18, more sequestra removed.

May 8, excellent condition, consolidation advanced.

CASE XI.—A. K., aged thirty-two years, wounded September 6 by bursting shell in the thigh and gunshot wound of shoulder. Entered hospital September 14.

Fracture of right femur, chronic suppuration, transversal drain. Consolidation in an angle with apex pointing outward.

January 28, reduction, curettement of focus, removal of two sequestra. Plaster waistband and two collars joined by two tin strips.

Discharged February 26 in good condition.

CASE XII.—E. F., aged twenty-eight years, wounded January 29. Entered hospital January 30.

Fracture of right leg, both bones involved, with complete destruction to the extent of five to six centimetres. Removal of the bone splinters.

Application of plaster collars and three tin strips. Pain relieved at once.

CASE XIII.—A. L., aged twenty-five years, wounded January 22 by bursting shell. Entered hospital February 2.

Fracture of right arm. Bullet entered at the lower third of arm and made its exit in the upper third, resulting in a "seton wound" of the soft structures. Suppuration, fracture of humerus without splinters.

Incision, cleaning, and extraction of a bit of projectile the size of a pea.

Application of a plaster collar around the neck and shoulder, and another around the arm, these being joined by two tin strips. Frequent painless dressings. Patient discharged on February 22 in excellent condition.

CASE XIV.—J. L., aged thirty-two years, wounded February 6 by bursting shell. Entered hospital February 7.

Fracture of right tibia, penetrating wound just below the knee.

Fracture focus cleaned and all tissue not completely detached was left in place. Immobilization by two plaster collars, one surrounding the thigh, the other the lower part of the leg, these being joined by two tin strips.

February 17, extensive suppuration in the deep spaces of the thigh; incision, drainage.

March 7, supracondyle amputation.

CASE XVI.—L., wounded May 12 in the right upper limb by shrapnel. There was a traumatism with a single opening in the

antero-external aspect of the upper third of the thigh, which it did not appear to penetrate.

On the antero-external surface of the lower third were two wounds on the same vertical line, four fingers' breadth from each other, with infiltration, inflammatory reaction, and pain in the parts. On the posterior aspect were two orifices corresponding to those on the anterior aspect of the thigh. Fracture of the bones of the lower end of the leg and foot; parts infiltrated.

May 17, incision uniting the two wounds in the antero-external aspect and splitting up of the aponeurosis. A large focus of pus was thus exposed between the tibia and fibula. Behind, an incision was made, uniting both wounds there. The limb was immobilized in plaster collars joined by tin strips.

Patient discharged in good condition on October 14.

CASE XVII.—B., wounded May 23. Great destruction of tissue, with multiple wounds of the lower end of the left forearm. Application of plaster collars and tin strips. The necessary frequent dressings were painless.

CASE XVIII.—J., wounded September 25 by bursting shell, bits of which entered the right leg in the area of the tendo achillis and made their exit in front of the internal malleolus.

Bone splinters could be felt in the area of the malleolar wound. The malleolus was fractured, but the joint did not appear to have been opened. Application of plaster collars and tin strips. Result excellent.

CASE XIX.—B., comminuted fracture of the middle third of the leg by bursting shell. Wound laid open by incision, extraction of bone splinters. Plaster collars and tin strips, which relieved all pain. Recovery excellent.

CASE XX.—J. N., aged thirty-five years, wounded September 14.

Compound fracture of the middle third of right leg. Large wound, 15 centimetres long, occupying the entire antero-external aspect of the leg. The muscles of the antero-external space were reduced to pulp, forming a large hernia. In the midst of the wound the fragments of the tibia protruded. They were irregular and greatly separated. Enormous bone splinters were removed before the patient was transferred to hospital. The fibula was in the same condition.

The lower portion of the leg was simply connected with the remainder by the soft structures of the posterior portion. However, the foot was alive, the circulation being sufficient. The tendons of the back of the foot were without action, and the toes were flexed. The general position of the foot was in varus. Patient's condition low, temperature 40° C., rapid, irregular pulse, chills, insomnia, etc.

After a long and thorough cleaning of the fracture focus, three plaster collars were applied, one surrounding the foot and three connecting tin strips.

October 18, condition much better, wound healthy red, temperature 38° C.

CASE XXI.—B. P., aged twenty-eight years, wounded September 13 in the knee and right elbow by bursting shell.

Typical resection of knee and plaster collars with arches of tin strips applied. The collars were broad and included the thigh and leg.

Fracture of the epiphysis of the humerus. Three plaster collars, one on the arm joined to the other two by tin strips.

Both the arm and leg could be moved and dressed without the slightest difficulty.

CASE XXII.—J. B., aged thirty-seven years, wounded July 1. Fracture of right leg and foot from bursting shell.

Application of plaster collars and tin strips.

Discharged in excellent condition October 5.

CASE XXIII.—A. M., aged twenty-five years, wounded June 6. Large wound at the internal aspect of the lower part of the right thigh over Hunter's canal. Gas in the tissues. Comminuted fracture of femur. Two plaster collars, one on the thigh, the other on the leg, joined by tin strips. Dressing painless. General condition good.

CASE XXIV.—J. M., aged twenty-three years, wounded March 18 by bursting shell. Entered hospital March 30.

Irregular wound of right leg. Comminuted fracture of both bones at their lower third. Plaster collars and tin strips. From the time of their application the patient no longer suffered.

October 9, patient walked well with a straight leg.

CASE XXV.—J. L., aged twenty-two years, wounded June 6 by a bursting shell. Entered hospital June 18.

Right hand shattered. The lower end of the ulna and the external aspect of two rows of carpal bones were exposed. Cleaning of wound. Plaster collars on the palmar side with tin strips. Small plaster collars on the forearm and fingers.

October 9, condition excellent, good consolidation.

CASE XXVI.—J. O., aged twenty-four years, wounded June 16 by shrapnel. Entered hospital June 21.

Multiple wounds on dorsal aspect of the end of the right forearm and arm. There was a large loss of substance in the right thigh and extensive sloughing. Fracture at the lower third of the femur. There was a deep penetrating wound of the right foot, with lesions of the scaphoid and astragalus.

June 21, the wounds were curetted and the parts put up in plaster cases. This was repeated on June 27.

July 13, a Delbet's apparatus was applied to the thigh. On July 17 a plaster case was applied to the foot.

October 9, general condition good. Walked. Only a very slight shortening. Rectitude was sufficient.

CASE XXVII.—G. M., aged twenty-two years, wounded August 13. Penetrating wound of left knee. Bilateral arthrotomy. Plaster collars above and below knee, joined by tin strips.

October 9, the patient was up and about in good condition.

In all the above cases, which were under my observation until complete cure, and where amputation was not required, it will be seen that the orthopaedic result was good and the functions were restored.

The cases in which the plaster collars and tin strip splints find their special indications are, first, those which give rise to intense pain.

In great joint wounds involving the knee or elbow their use is particularly happy, the collars being very broad, including the thigh and leg, the arm and forearm. In the latter case curved tin strips maintain the forearm at any desired angle on the arm. Open fractures of the leg, thigh, and forearm are greatly benefited by their use.

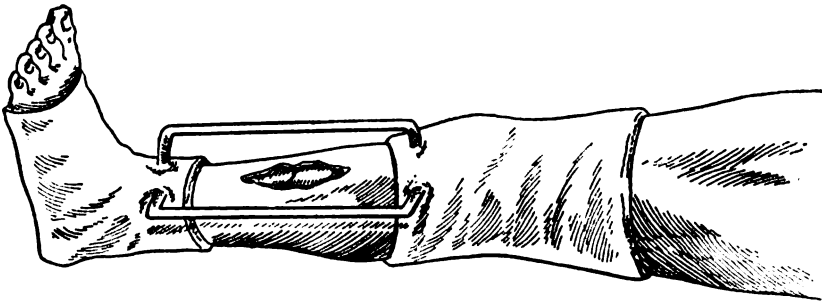
The plaster collars are merely segments made with ordinary plaster incorporated in gauze of various breadths. For fractures of the thigh a plaster waistband with an under-thigh extension is applied. For those of the arm, a collar should be applied around the neck and shoulder.

Bands of tarlatan about one and a half metres and six thicknesses are cut in breadths of 15, 10, and 6 centimetres. The tin strips should be made from galvanized tin, 27 to 20 millimetres thick. Galvanized tin should always be used.

At the time of application the plaster is dipped for a minute in water and the ordinary technic employed as for any plaster cast.

Let us take as an example the application of an apparatus destined to immobilize the knee in the case of a large joint wound. The leg being raised by one or two assistants, a very broad collar is first placed on the thigh, using the 10- or 15-centimetre bands and rolled in such a way that the minimum thickness is not less than three bands; that is to say, 18 layers of tarlatan.

FIG. 2



Open fracture of leg. Plaster collars and metal splints applied.

A broad collar is next put on the leg, leaving the knee completely free between the two.

Should the plaster bands be applied directly on the skin? Usually the patients do not complain when this is done on the condition that they are *laid on and not tight*. They do not give rise to œdema, ulceration, or sloughing, but it is always better to separate the plaster from direct contact with the skin by placing a very thin sheet of cotton batting over the parts.

When the first layer of plaster rollers have been applied the tin strips are next put in place. They should have been previously cut and curved approximately to the shape of the limb, and it is more prudent to cover their ends with some tightly rolled tarlatan, which is then dipped in plaster so that they may be better incorporated in the collars.

The curves must be very carefully corrected to the shape of the limb, and when this is done three more thicknesses of roller bandages (six thicknesses of gauze each) are applied and allowed to dry.

It is clear that the above technic will vary only in details and will retain the above general principle in all cases, no matter what may be the shape and extent of the apparatus.

Certain criticisms have been made regarding this type of splint, the principal one being that immobilization is imperfect, thus allowing the osseous fragments to assume faulty positions from muscular action, resulting in pseudarthroses and functional impotency.

Now, from the case reports given, it is evident that these fears are unfounded. But it must be said that, no matter how simple the application of these splints is, the theory of their function should be thoroughly understood. For example, an apparatus composed entirely of tin arches is to be avoided, especially if the collars are situated far apart. These should be used only for the area of the wound, so that it can be easily exposed to view and facilitate access to it, while at other points straight tin strips are to be used, which can be made to mould themselves on the limb and thus retain it in proper position.

When the site of the wound will permit, the strips should always be straight. Some experience is, of course, necessary to apply the plaster collars without too much pressure and still sufficiently close to the limb to prevent it from moving in the collar. When continued extension is to be used, the collars must be given a point of application on the prominent portions of the limb and surround as long a segment of the member as possible.

Another defective point in this type of splint, say the critics, is that they require watching. Now, I am unaware of any form of splint that does not necessitate careful oversight, and, just as in other types of fracture apparatuses, if the temperature goes up, and this can not be explained by the parts that are visible or accessible to inspection, one must ascertain that sloughing is not under way at some point hidden by the collars, or if an extension of the purulent tracts is taking place under them. But it is precisely from the fact that the wounds can be better watched with this form of splint that such possibilities are far less likely to happen.

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